39700-5

IN THE COURT OF APPPEALS FOR THE STATE OF WASHINGTON
DIVISION II

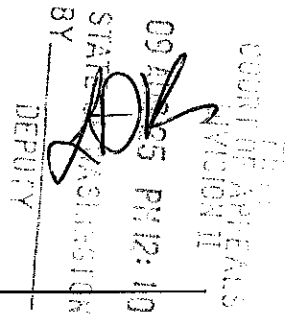
IN RE THE PERSONAL RESTRAINT PETITION OF:

EDWARD M. GLASMANN,

PETITIONER.

84475-5

PERSONAL RESTRAINT PETITION



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A. STATUS OF PETITIONER

Edward M. Glasmann (hereinafter "Glasmann") challenges his Pierce County convictions for kidnapping, assault, attempted robbery, and obstructing and the subsequently imposed 210 month sentence (Pierce County Case No. 04-1-04983-2).

Mr. Glasmann is currently confined at the Washington State Reformatory in Monroe, Washington. This petition, filed within one year of the September 11, 2008 mandate from Glasmann's direct appeal, is his first collateral attack on his judgment of conviction.

B. FACTS

Procedural History

On October 24, 2004, Mr. Glasmann was charged by Information with several crimes arising from an incident occurring two days earlier—on October 22nd. After numerous continuances and changes in counsel, Glasmann eventually proceeded to jury trial in April-May 2006. Glasmann's jury convicted him of second-degree assault, first-degree kidnapping, attempted second-degree robbery, and obstructing. He was subsequently sentenced to a total of 210 months in prison.

Mr. Glasmann appealed. His conviction was affirmed on appeal. This Court's decision was issued on January 23, 2008. Glasmann then unsuccessfully sought review by the Supreme Court. The mandate was issued on September 11, 2008.

Facts

This Court described the facts on direct appeal as follows:

Edward Michael Glasmann and Angel Benson were romantically involved and engaged to be married. On the night of October 22, 2005, Glasmann and Benson went to dinner in Tacoma and rented a motel room in Lakewood to celebrate Glasmann's birthday. Both Glasmann and Benson ingested methamphetamine, ecstasy, and alcohol over the course of the evening. In addition, Glasmann and Benson had been arguing throughout that day and evening.

Around midnight, their argument escalated. Glasmann hit Benson, who curled up into the fetal position to protect herself from his blows. Glasmann eventually told Benson that he wanted to go for a ride. They both left the motel room.

Outside the room, another hotel guest, Erika Rusk, witnessed Glasmann (1) pin Benson against the wall with one hand around her neck and repeatedly punch her with his other hand; (2) release Benson and kick her twice in the stomach; (3) drag her to the passenger side of his Corvette and got into the driver's seat; (4) reach over to the open passenger door and attempt to pull Benson into the car by her hair; (5) pull forward from the parking stall while Benson was not fully in the car; and (6) run over Benson's leg with his car.

Once in the car, Benson put the car into park, grabbed the keys, and ran into a mini-mart adjacent to the motel. Inside the mini-mart, she hid on the floor behind the counter. As Rusk watched, she was calling 911 and reporting these events to dispatch.

Lakewood Police Officers Timothy Borchardt and David Butts arrived to find Glasmann's Corvette parked in the roadway. As they approached, they observed Glasmann exit his Corvette, run over to the mini-mart, and climb into three separate cars, apparently hoping to steal one and escape.

Their guns drawn, Officers Borchardt and Butts ordered Glasmann to show his hands. Glasmann refused to comply, and told the officers that he had a gun. When Glasmann pushed a man aside in order to access the third car, Officer Butts approached the open driver's side window and sprayed pepper spray into Glasmann's eyes. Glasmann then exited the vehicle through the passenger door and ran into the

mini-market, pursued by a group of officers.

> Glasmann continued to yell, "[S]hoot me, I have got a gun. Go ahead and shoot me." 4 Report of Proceedings (RP) at 116. As if it were a weapon, he pointed a black object at the officers. Eventually, Glasmann ran behind the counter, grabbed Benson, put his arm around her neck in a choke hold, and pulled her body in front of his, threatening to kill her. Glasmann then dropped to the floor, holding Benson between him and the officers.
>
> When Benson was able to "wiggle her way down from [Glasmann's] body," Officer Ryan Hamilton applied a stun gun to Glasmann. 4 RP at 125-26. The officers then removed Benson. They took Glasmann into custody, determined he was not armed, and realized he had brandished a stereo remote control as a weapon.

Facts that either were not introduced or discovered at the time of trial, relevant to this PRP, are both appended to this PRP and are discussed in the pertinent claims of error below.

C. ARGUMENT

1. MR. GLASMANN WAS DENIED HIS SIXTH AMENDMENT RIGHT TO EFFECTIVE ASSISTANCE OF COUNSEL WHEN COUNSEL FAILED TO ELICIT TESTIMONY OF GLASMANN'S IMPAIRMENT AT THE TIME OF THE CRIME—TESTIMONY THAT WOULD HAVE MERITED A VOLUNTARY INTOXICATION INSTRUCTION.

On direct appeal in his statement of additional Grounds (SAG) Glasmann contended his attorney rendered ineffective assistance of counsel by failing to request an intoxication instruction. This Court rejected that argument, concluding:

> ....the record does not contain ample evidence that his level of intoxication affected his ability or lack thereof to form the mental state required to establish the crimes charged. At best, the evidence merely showed that Glasmann had ingested unspecified amounts of

> methamphetamine, ecstasy, and alcohol the night of the incident. As such, Glasmann was not entitled to an involuntary intoxication instruction.

(internal citation removed).

Glasmann now supplies sufficient "evidence that his level of intoxication affected his ability or lack thereof to form the mental state required to establish the crimes charged." The evidence presented here was available to defense counsel had counsel either conducted a competent investigation or elicited the testimony from Petitioner. Given that Glasmann's ingestion of mind altering substances was known to his jurors, coupled with the defense focus on his state of mind, there was no tactical reason to fail to present a case justifying a voluntary intoxication instruction. In any event, Glasmann has certainly presented sufficient evidence for this Court to remand this claim to the trial court for an evidentiary hearing. RAP 16.11.

At trial, Glasmann testified he was "high" on methamphetamine and ecstasy. 9 RP 367-69. However, trial counsel did not delve into this issue deeper—likely due to his failure to sufficiently investigate, including failing to sufficiently meet and speak with Glasmann before trial

Mr. Glasmann has a long, unfortunate history of drug and alcohol abuse. Due to his excessive and chronic use of substances, Glasmann has repeatedly suffered from blackouts. In addition, he suffers from extreme emotional swings and paranoia, common side effects of substance

addiction. *See Declaration of Glasmann.*

During the month leading up to these events, Glasmann was "using between 7 and 14 grams of methamphetamine every day." *Id.* As a result, Glasmann "went without sleep for many days," became "extremely paranoid and agitated," had "episodes of broken reality and suicidal thoughts and plans of suicide," and would often "blackout." *Id.* As of the day of the crime, Glasmann had been "abusing methamphetamine every day for over 2 months." He used an astonishing "10 grams of (m)ethamphetamine during the day" of the incidents, then added six to ten drinks and ecstasy on top of that drug use. *Id.* However, that was not the extent of Glasmann's drug use. After arriving at a hotel with Ms. Benson, Glasmann used "2-3 grams" of methamphetamine, in addition to the aforementioned ecstasy. *Id.*

This evidence, if introduced, would have been sufficient to support a voluntary intoxication instruction. However, there was more.

The effect of this massive ingestion of mind altering substances is evident from evidence both introduced at trial and available to defense counsel, but not introduced for reasons unknown. *See Declaration of Glasmann* (Defense counsel "told me he was going to introduce these facts into trial and that it would be the primary defense," but "did not discuss any investigation work with me and I am not aware of any investigations conducted." "I was never presented with any information regarding how

the drugs and alcohol use affected my state of mind and we never discussed it further after deciding that we would use it in the defense.").

Perhaps most astonishing is the evidence that a police officer responding to the scene at the convenience store "repeatedly stomped" on Glasmann's head "with his boot while I was being held down," a fact that Glasmann cannot recall. Glasmann's lack of recall of that incident certainly supports his diminished capacity due to intoxication for the criminally charged events. *See* Appendix B.

Ms. Benson, the victim, likewise could have provided additional support. In her subsequent civil deposition, she noted a "Jekyll and Hyde" transformation that came with Glasmann's use of methamphetamine, along with at least one incident of prior blackout behavior. *See* Appendix C.

It would have been easy to support this testimony with an expert opinion about the effects of this constellation of substances, especially the methamphetamine. *See* Appendix E – G. Methamphetamine increases the release and blocks the reuptake of the brain chemical (or neurotransmitter) dopamine, leading to high levels of the chemical in the brain, a common mechanism of action for most drugs of abuse. Dopamine is involved in reward, motivation, the experience of pleasure, and motor function. Methamphetamine's ability to rapidly release dopamine in reward regions of the brain produces the intense euphoria, or "rush," that many users feel after snorting, smoking, or injecting the drug. Appendix E.

Methamphetamine enters the brain quickly and lingers longer than other similar drugs of abuse. Appendix F.

Most importantly, methamphetamine is highly correlated with "severe neurologic and psychiatric adverse events, including the development of psychotic states." Appendix G.

There certainly was ample evidence suggesting that Glasmann was psychotic at the time of the crime. He was largely impervious to pepper spray and a taser. However, without the necessary context and supporting evidence, these facts alone were insufficient for a voluntary intoxication instruction, if one had been requested. However, adding the new evidence, available if only a minimally competent investigation had been undertaken, the evidence is clearly sufficient—not only for an instruction, but which raises a reasonable likelihood of a different outcome on at least one of the counts of conviction.

In short, evidence was available that would have supported a viable defense of voluntary intoxication. As this Court laid out on direct appeal, a criminal defendant is entitled to a voluntary intoxication instruction if: (1) one of the elements of the crime charged is a particular mental state; (2) there is substantial evidence of ingesting an intoxicant; and (3) the defendant presents evidence that this activity affected his ability to acquire the required mental state. *State v. Harris,* 122 Wn.App. 547, 552, 90 P.3d 1133 (2004). In other words, the evidence must reasonably and logically

connect Glasmann's intoxication with his asserted inability to form the requisite level of culpability to commit second degree assault. *See State v. Griffin,* 100 Wn.2d 417, 418-19, 670 P.2d 265 (1983); *State v. Kruger,* 116 Wn.App. 685, 692, 67 P.3d 1147 (2003) (stating that mere intoxication is not enough; rather, the evidence must show the effects of the intoxicant).

Although an expert opinion would have been most helpful (as is discussed in the next section), if counsel had conducted even an expert-less investigation and presented the available evidence, he could have obtained an instruction. But "[i]f the issue involves a matter of common knowledge about which inexperienced persons are capable of forming a correct judgment, there is no need for expert opinion." *State v. Smissaert,* 41 Wash.App. 813, 815, 706 P.2d 647 (1985). And "[c]ertainly the effects of alcohol upon people are commonly known and all persons can be presumed to draw reasonable inferences therefrom." *Id.* So where the court gives a voluntary intoxication instruction, the defendant can argue diminished capacity without an expert. *Id.*

Effective assistance of counsel is guaranteed by the Sixth Amendment to the United States Constitution. *Strickland v. Washington,* 466 U.S. 668 (1984). To establish that trial counsel's representation was constitutionally inadequate, Glasmann must show that counsel's performance was deficient—*i.e.*, that it fell below an objective standard of reasonableness—and that the deficient performance was prejudicial.

Strickland, 466 U.S. at 687-88. The proper measure of attorney performance is reasonableness under prevailing professional norms. *Id.* at 688. In order to demonstrate prejudice arising from counsel's deficient performance, Glasmann must show that there is a reasonable probability that, but for counsel's errors, the result of the proceeding would have been different. *Strickland,* 466 U.S. at 694.

A reasonable probability is a probability sufficient to undermine confidence in the outcome. *Id.* The "reasonable probability" standard is not stringent, and requires a showing by less than a preponderance of the evidence that the outcome of the proceeding would have been different had the claimant's rights not been violated. *See, e.g., Pirtle v. Morgan*, 313 F.3d 1160, 1172 (9th Cir. 2002), *cert. denied*, 539 U.S. 916 (2003), quoting *Strickland*, 466 U.S. at 694:

> A "reasonable probability" is less than a preponderance: "the result of a proceeding can be rendered unreliable, and hence the proceeding itself unfair, even if the errors of counsel cannot be shown by a preponderance of the evidence to have determined the outcome."

In recent years, trial counsel's duty to thoroughly investigate, prior to making reasonable trial tactic calculations, has been clearly defined. *See, e.g., Rompilla v. Beard,* 545 U.S. 374 (2005); *Wiggins v. Smith*, 539 U.S. 510 (2003); *Williams v. Taylor*, 529 U.S. 362, 395-96 (2000). These three

cases applied the *Strickland* rule—that counsel must conduct a competent investigation before making tactical choices.

In this case, defense counsel was aware of Glasmann's intoxication prior to trial. In fact, he suggested to Glasmann that intoxication would be the focus of the defense. However, trial counsel failed to further investigate and, as a result, failed to present Glasmann's available evidence. In fact, trial counsel failed to request an instruction supporting what he told Glasmann was the chosen defense.

As a result, Glasmann's jury was left with evidence of illegal drug use, but no vehicle to apply it to diminish Glasmann's legal responsibility. In other words, trial counsel drew out all of the prejudicial nature of drug use, but sought none of the potential legal benefit. Washington courts have acknowledged that evidence of drug use—standing alone—is highly prejudicial. "in view of society's deep concern today with drug usage and its consequent condemnation by many if not most, evidence of drug addiction is highly prejudicial in the minds of the average juror." *State v. Renneberg,* 83 Wn. App. 735, 737, 522 P.2d 835 (1974). Thus, aside form laying the ground-work for a voluntary intoxication defense, the voluntary introduction of drug use cannot be pinned to any legitimate strategic consideration. Without any testimony to explain the impact of the drug use on his state of mind, accompanied by an instruction allowing a jury to give effect to that condition, trial counsel left Glasmann's jury with the solid

impression that he was simply a "law breaking drug user." *See State v. Powell,* 139 Wn. App. 808, 162 P.3d 1180 (2007).

Aided by an evidentiary hearing (and the right to expert assistance that accompanies it pursuant to RAP 16.12), Glasmann will be able to thoroughly establish that his level of intoxication and its effect on his state of mind would have supported a voluntary intoxication instruction—evidence that must undermine confidence in the verdicts.

Thus, if the State contests Glasmann's evidence in this claim, this Court should remand for an evidentiary hearing. RAP 16.11. Otherwise, this Court should grant Glasmann's petition and remand for a new trial.

2. MR. GLASMANN WAS DEPRIVED OF HIS SIXTH AMENDMENT RIGHT TO EFFECTIVE ASSISTANCE OF COUNSEL WHEN COUNSEL FAILED TO INVESTIGATE AND CALL AN EXPERT WITNESS WHO COULD HAVE GIVEN TESTIMONY CONNECTING GLASMANN'S EXTREME INTOXICATION WITH HIS CORRESPONDING ABILITY, OR LACK OF ABILITY, TO FORM THE CRIMINAL INTENT REQUIRED FOR CONVICTION.

Although some of the details were disputed, Mr. Glasmann did not dispute the essential characterization of the facts that lead to his arrest and eventual convictions. The dispute was over the degree of crimes committed. Counsel sought and Glasmann's jury was instructed on lesser included offenses. In fact, counsel affirmatively argued that the jury find Glasmann guilty of the lesser offenses. In short, Glasmann's state of mind at the time of the crime was critical to the defense theory—in fact, it was the only issue.

The charge that carried the greatest penalty, kidnapping in the first degree is a specific intent crime. Defense counsel asked jurors to find Glasmann guilty of the lesser crime of unlawful imprisonment. 8 RP 494.

Given this backdrop, counsel's failure is astonishing. Nothing could have been more critical to the case than for the jury to have been presented with information about the affect of Glasmann's drug ingestion on his state of mind at the time of the crime.

There was ample evidence available to counsel to establish Glasmann's high level of intoxication, appoint discussed in the previous claim of error. Counsel elicited testimony from Glasmann that he was "out of my head" on drugs. 6 RP 379. Glasmann was impervious to the pepper spray. He was acting erratically. He spoke nonsense.

Ms. Benson testified that Glasmann ingested methamphetamine. She stated that his driving was so impaired she was afraid of a wreck. She described him as suffering from "raod rage." 3 RP 87-88. She told a defense investigator that Glasmann was "weird that night and not himself." 3 RP 147.

Counsel even began closing argument with references to the drug use. However, inexplicably, counsel failed to conduct a competent investigation and failed to call an expert witness to testify about Glasmann's impaired state of mind.

As indicated earlier, there is no shortage of documentation available

to establish the drastic effect of methamphetamine on the brain. The drug, even in low to moderate amounts, causes anxiety, confusion, and mood disturbances and can lead to violent behavior. With chronic use and/or the consumption of larger doses, methamphetamine causes psychotic features, including paranoia, visual and auditory hallucinations. *See* Appendix E, F, G.

All of these disturbing consequences were on display on the night of these crimes. As a result, it was deficient for trial counsel not to consult with an expert. Indeed, that expert would not necessarily need to assess Mr. Glasmann. It would have been sufficient, although not preferable, to discuss the impact of the amount of drugs ingested on the human central nervous system.

Counsel must, at a minimum, conduct a reasonable investigation enabling him to make an informed decision about how best to represent his client. *In re Pers. Restraint of Brett,* 142 Wn.2d 868, 873, 16 P.3d 601 (2001); *Seidel v. Merkel,* 146 F.3d 750, 755 (9th Cir. 1998).

If investigated, the evidence would have been admissible. Kidnapping is a specific intent crime. Thus, evidence of an inability to form the requisite intent is admissible. *State v. Martin,* 14 Wn. App. 74, 538 P.2d 873 (1975). Intoxication is an accepted basis for arguing lack of ability to form the requisite intent. RCW 9A.16.090. The right to present evidence includes the right to expert testimony.

A case particularly on point is *Miller v. Terhune,* 510 F. Supp.2d 486 (E.D. Cal. 2007). The central issue before the court in that case was whether petitioner's trial counsel rendered ineffective assistance when they failed to investigate and present evidence as to how petitioner's level of intoxication likely affected his perceptions, intentions and actions on the night of the shooting.

After finding that counsel failed to conduct a minimally competent investigation (counsel did consult with mental health professionals, unlike this case), the Court held:

> In sum, the record reflects that counsel failed to investigate the effects of intoxication on petitioner. Accordingly, counsel was in no position to make a reasoned or strategic decision regarding the use of intoxication evidence. It is well settled that under *Strickland,* "attorneys have considerable latitude to make strategic decisions about what investigations to conduct once they have gathered sufficient evidence upon which to base their tactical choices." *Jennings v. Woodford,* 290 F.3d 1006, 1014 (9th Cir.2002). In the instant case, there is simply no indication that defense counsel gathered any evidence upon which to base their decision to not investigate or present evidence of intoxication. *See Williams,* 529 U.S. at 369, 120 S.Ct. 1495(counsel must conduct a "thorough investigation" before decision can be considered strategic under *Strickland*); *Sanders,* 21 F.3d at 1457 (citing *United States v. Gray,* 878 F.2d 702, 711 (3rd Cir.1989)) (finding that "... [c]ounsel can hardly be said to have made strategic choice when he has not obtained the facts on which a decision could be made.").
>
> The ABA Standards for Criminal Justice provide guidance as to the obligations of criminal defense attorneys in conducting an investigation. *Rompilla v. Beard,* 545 U.S. 374, 125 S.Ct. 2456, 162 L.Ed.2d 360 (2005); *Williams,* 529 U.S. at 396, 120 S.Ct. 1495. The standards in effect at the time of petitioner's trial clearly described the defense lawyer's duty to investigate:

> (a) Defense counsel should conduct a prompt investigation of the circumstances of the case and explore all avenues leading to facts relevant to the merits of the case and the penalty in the event of conviction. The investigation should include efforts to secure information in the possession of the prosecution and law enforcement authorities. The duty to investigate exists regardless of the accused's admissions or statements to defense counsel of facts constituting guilt or the accused's stated desire to plead guilty.
>
> ABA Standards for Criminal Justice, Defense Functions, Standard 4-4.1 (3d Ed.).
>
> When trial counsel is on notice that his client may have a particular mental impairment relevant to the case, the Ninth Circuit has repeatedly held that failure to investigate the mental state constitutes deficient performance under *Strickland. See, e.g., Douglas v. Woodford,* 316 F.3d 1079, 1085 (9th Cir.2003) (citing *Bean v. Calderon,* 163 F.3d 1073, 1078 (9th Cir.1998) (holding that "[t]rial counsel has a duty to investigate a defendant's mental state if there is evidence to suggest that the defendant is impaired."); *see also Caro v. Woodford,* 280 F.3d 1247, 1254 (9th Cir.2002); *Hendricks v. Calderon,* 70 F.3d 1032, 1043 (9th Cir.1995)). In such circumstances, counsel must undertake at least "a minimal investigation in order to make an informed decision regarding the possibility of a defense based on mental health." *Seidel v. Merkle,* 146 F.3d 750, 756 (9th Cir.1998).

Id. at 499.

In *Jennings,* one of the cites cases, the 9th Circuit concluded that defense counsel was ineffective when counsel failed to investigate Jennings use of methamphetamine and alcohol on the night of the crime, despite the fact that Jennings was insistent on an alibi defense. The Court concluded that the decision to pursue the alibi defense was uniformed and therefore unreasonable. *Id.* at 1014.

Had defense counsel in this case simply looked to the relevant

caselaw, he would have discovered *State v. Kruger, supra,* a case with remarkably similar facts. *Id.* at 692. ("The record reflects substantial evidence of Mr. Kruger's drinking and level of intoxication. And there is ample evidence of his level of intoxication on both his mind and body, e.g., his "blackout," vomiting at the station, slurred speech, and imperviousness to pepper spray. He was entitled to the instruction.").

Had counsel conducted a simple search of the caselaw for "methamphetamine" and "mental state," he would have found a plethora of cases discussing, with approval, the use of an expert to opine about the interaction of drugs on the brain. *See e.g., State v. Ferrick,* 81 Wn.2d 942, 944, 506 P.2d 860 (1973); *State v. Coates,* 107 Wn.2d 882, 735 P.2d 64 (1987); *State v. Griffin,* 100 Wn.2d 417, 419, 670 P.2d 265 (1983); *State v. Hansen,* 46 Wn. App. 292, 730 P.2d 706 (1987); *State v. Thomas,* 109 Wn.2d 222, 743 P.2d 816 (1987); *State v. Cienfuegos,* 144 Wn.2d 222, 25 P.3d 1011 (2001).

Thus, Glasmann has presented at least a *prima facie* claim of error. As before, he is entitled to an evidentiary hearing or reversal, if the State does not dispute these facts with their own competent evidence.

3. MR. GLASMANN WAS DENIED HIS SIXTH AMENDMENT RIGHT TO EFFECTIVE ASSISTANCE OF COUNSEL WHEN COUNSEL INEXPLICABLY OPENED THE DOOR TO THE ENTIRETY OF GLASMANN'S CRIMINAL HISTORY.

Prior to trial, the court granted an *in limine* motion limiting the

impeachment of Glasmann to a conviction for unlawful issuance of a bank check and a second-degree robbery. 1 RP 5-6.

When Glasmann was on the stand, counsel asked him if he had 'ever been convicted of a crime before" to which Glasmann responded by admitting to the convictions earlier allowed by the court. 6 RP 387-88.

In response, the State argued and the court agreed the door had been opened by the phrasing of counsel's question. Thus, the State was able to elicit numerous other crimes—making Glasmann out to be both a career criminal and a liar.

This is a very straightforward case of attorney incompetence. It was obviously not tactical—counsel argued that he did not open the door. Thus, the deficient performance prong is easily satisfied.

Prejudice, which is defined as undermining confidence in the verdict, is also established. Washington caselaw is clear that "prior conviction evidence is inherently prejudicial when the defendant is the witnesses because it tends to shift the jury focus form the merits of the charge to the defendant's general propensity for criminality." *State v. Jones,* 101 Wn.2d 113, 120, 677 P.2d 131 (1984), *overruled on other grounds State v. Brown*, 111 Wn.2d 124, 761 P.2d 588 (1988).

However, this Court must add to the "inherent" prejudice, the State's exploitation of this error. During closing argument, discussed more fully in the next section, the State repeatedly and unfairly) attacked Glasmann's

honesty. Thus, this serious misstep by counsel inflicted a great blow on the jury's assessment of Glasmann. If he was willing to lie by minimizing his criminal history, he was also likely not honest in describing his own involvement in the events at issue. In the end, counsel's blunder injured Glasmann's credibility more than any cross-examination could.

Glasmann has established his claim of ineffective assistance of counsel. He is entitled to a new trial.

4. MR. GLASMANN WAS DEPRIVED OF DUE PROCESS AND THE RIGHT TO A FAIR TRIAL WHEN THE PROSECUTOR USE HIGHLY INFLAMMATORY ILLUSTRATIONS AND INJECTED PERSONAL OPINIONS DURING CLOSING ARGUMENT

During closing argument, the prosecutor presented a PowerPoint slide show to the jury. Those slides are attached. *See* Appendix H, obtained through a public disclosure request). The slides, which speak for themselves, show photos of Glasmann's bruised "mug shot" face (as a result of being stomped by a police officer's boot), accompanied by the headings: "Do You Believe Him;" "Why Should You Believe Anything He Says About the Assault?;" and the word "GUILTY" superimposed on his face.

Not only are the booking photos unfair, the prosecutor repeated expressed his personal opinion about the credibility of witnesses—a decision that must be left in the exclusive hands and minds of jurors.

In order to establish prosecutorial misconduct, a defendant must

prove that the prosecutor's conduct was improper and that it prejudiced his right to a fair trial. *State v. Carver,* 122 Wash.App. 300, 306, 93 P.3d 947 (2004) (citing *State v. Dhaliwal,* 150 Wash.2d 559, 578, 79 P.3d 432 (2003)). A defendant can establish prejudice only if there is a substantial likelihood that the misconduct affected the jury's verdict. *Carver,* 122 Wash.App. at 306, 93 P.3d 947 (quoting *Dhaliwal,* 150 Wash.2d at 578, 79 P.3d 432). Courts review a prosecutor's comments during closing argument in the context of the total argument, the issues in the case, the evidence addressed in the argument, and the jury instructions. *Carver,* 122 Wash.App. at 306, 93 P.3d 947 (citing *Dhaliwal,* 150 Wash.2d at 578, 79 P.3d 432). If defense counsel fails to object to the prosecutor's statements, then reversal is required only if the misconduct was so flagrant and ill-intentioned that no instruction could have cured the resulting prejudice. *State v. Belgarde,* 110 Wash.2d 504, 508, 755 P.2d 174 (1988).

It is improper for a prosecutor to personally vouch for a witness's credibility. *See State v. Brett,* 126 Wash.2d 136, 175, 892 P.2d 29 (1995), *cert. denied,* 516 U.S. 1121, 116 S.Ct. 931, 133 L.Ed.2d 858 (1996). Prosecutors may, however, argue an inference from the evidence and this court will not find prejudicial error "unless it is 'clear and unmistakable' that counsel is expressing a personal opinion." *Brett,* 126 Wash.2d at 175, 892 P.2d 29 (quoting *State v. Sargent,* 40 Wash.App. 340, 344, 698 P.2d 598 (1985)).

This Court views the "prejudicial or inflammatory effect" of the improper remarks "in context with the earlier evidence and the circumstances of the trial in which they were made." *State v. Green,* 71 Wn.2d 372, 381, 428 P.2d 540 (1967).

In this case, read in context, and especially considering the visual display accompanying the prosecutor's words, the argument was both inflammatory and unfair. Although Petitioner could make nuanced arguments about the impropriety of the argument, the picture of a bruised face of Glasmann accompanied by the superimposed word "GUILTY" says it all. This Court should condemn such unfair argument.

5. MR. GLASMANN IS ENTITLED TO A NEW TRIAL BASED ON THE CUMULATIVE PREJUDICE FROM MULTIPLE ERRORS, ESPECIALLY THE MULTIPLE FAILURES OF DEFENSE COUNSEL.

Where the cumulative effect of multiple errors so infected the proceedings with unfairness a resulting conviction or death sentence is invalid. *See Kyles v. Whitley,* 514 U.S. 419, 434-35, 115 S. Ct. 1555, 131 L. Ed.2d 490 (1995). As the Ninth Circuit pointed out in *Thomas v. Hubbard,* 273 F.3d 1164 (9th Cir.2001), "[i]n analyzing prejudice in a case in which it is questionable whether any single trial error examined in isolation is sufficiently prejudicial to warrant reversal, this court has recognized the importance of considering the cumulative effect of multiple errors and not simply conducting a balkanized, issue-by-issue harmless error review." *Id.* at 1178 (internal quotations omitted) (citing *United States*

v. Frederick, 78 F.3d 1370, 1381 (9th Cir.1996)); *see also Matlock v. Rose,* 731 F.2d 1236, 1244 (6th Cir.1984) ("Errors that might not be so prejudicial as to amount to a deprivation of due process when considered alone, may cumulatively produce a trial setting that is fundamentally unfair.").

Glasmann asserts that each of the errors described previously merits relief. However, considered cumulatively, they certainly resulted in sufficient prejudice to merit a new trial. Trial counsel's errors, measured cumulatively, were devastating to Glasmann. Counsel failed to investigate and present compelling evidence of the extent of Glasmann's intoxication, failed to obtain an instruction, opened the door to devastating evidence (making his client into a perjurer in the meantime), and failed to object and put an end to prosecutorial misconduct.

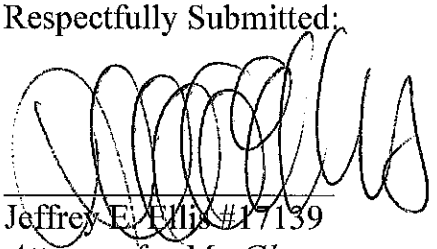
Glasmann is entitled to a new trial.

D. CONCLUSION AND PRAYER FOR RELIEF

Based on the above, this Court should either remand this case to Pierce County Superior Court for an evidentiary hearing or a new trial.

DATED this 21st day of August, 2009.

Respectfully Submitted:



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DO YOU BELIEVE HIM?

WHY SHOULD YOU BELIEVE ANYTHING HE SAYS ABOUT THE ASSAULT?

ASSAULT 1°

EVIDENCE
+
COMMON SENSE
+
LAW

GUILTY

ATTEMPTED ROBBERY 1°

INTENT TO COMMIT ROBBERY 1
+
SUBSTANTIAL STEP

GUILTY

KIDNAPPING 1°

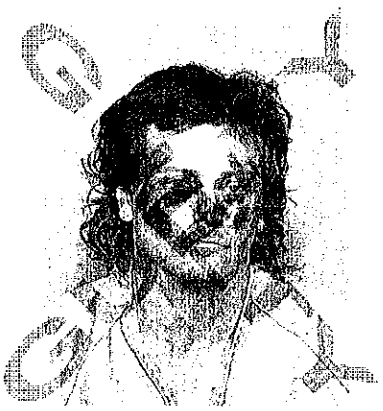
Intentionally abducted Angel Benson
+
With intent to use as shield or hostage

GUILTY

ATTEMPTED ROBBERY 2°

INTENT TO COMMIT ROBBERY 2
+
SUBSTANTIAL STEP

GUILTY



Appendix A

DECLARATION OF

EDWARD MICHAEL GLASMANN

I, Edward Michael Glasmann, declare the following:n

1. I started using Methamphetamine's in September of 1993.
2. I was clean for a period of time beginning in June of 2002
3. I started to use drugs again in January of 2004.
4. By July of 2004 I was addicted to methamphetamine and using every day.
5. From July 2004 to October 2004, I lost approximately 40 pounds.
6. During the month of October 2004, I was using between 7 and 14 grams of methamphetamines every day.
7. I commonly went without sleep for many days on end.
8. When using methamphetamines I was extremely paranoid and agitated.
9. I also had episodes of broken reality and suicidal thoughts and plans of suicide during my drug use.
10. After heavy methamphetamine use I would often blackout and lose time.
11. During the week prior to October 22nd, 2004, I was in two separate automobile accidents resulting from these blackouts.
12. On October 22nd, 2004 I had been abusing methamphetamine every day for over 2 months and had not had a full night's sleep in weeks.

13. October 22nd, 2004 was my 40th birthday and I was using drugs all day long. I had taken approximately 10 grams of Methamphetamine during the day prior to going to the casino at 8:00 pm.

14. I started drinking when I reached the casino. I had approximately six (6) to ten (10) drinks prior to leaving for the motel.

15. Around midnight Angel Benson and I rented a room at the Budget Inn Motel on South Tacoma Way.

16. When we went into the room we began using Methamphetamine and Ecstasy. I smoked and ingested approximately 2-3 grams of Methamphetamine and 1 tablet of Ecstasy.

17. Approximately 45 minutes later our argument escalated into a physical altercation and the events that I testified to at trial.

18. After my arrest I was assigned Raymond Thoenig as counsel.

19. Mr. Thoenig and I talked about a diminished capacity defense based upon my extreme intoxication.

20. Mr. Thoenig contacted a Psychologist, Brett Trowbridge, who spent one (1) hour with me in a consultation.

21. On May 17th, 2005 I was appointed John Cain as my counsel.

22. John Cain and I talked about a defense of diminished capacity and the drug use and blackout episodes.

23. Mr. Cain suffered from an aneurism and could not continue as my attorney.

24. On October 4th, 2005 was appointed Robert M. Quillian as my counsel.

DECLARATION OF

EDWARD MICHAEL GLASMANN

I, Edward Michael Glasmann, declare the following:

1. I started using methamphetamine in September of 1993.
2. I was clean for a period of time beginning in June of 2002
3. I started to use drugs again in January of 2004.
4. By July of 2004 I was addicted to methamphetamine and using every day.
5. From July 2004 to October 2004, I lost approximately 40 pounds.
6. During the month of October 2004, I was using between 7 and 14 grams of methamphetamine every day.
7. I commonly went without sleep for many days on end.
8. When using methamphetamine, I was extremely paranoid and agitated.
9. I also had episodes of broken reality and suicidal thoughts and plans of suicide during my drug use.
10. After heavy methamphetamine use I would often blackout and lose time.
11. During the week prior to October 22, 2004, I was in two separate automobile accidents resulting from these blackouts.
12. On October 22, 2004, I had been abusing methamphetamine every day for over 2 months and had not had a full night's sleep in weeks.
13. October 22, 2004, was my 40th birthday and I was using drugs all day long. I had taken approximately 10 grams of methamphetamine during the day prior to going to the casino at 8:00 pm.

14. I started drinking when I reached the casino. I had approximately six (6) to ten (10) drinks prior to leaving for the motel.

15. Around midnight Angel Benson and I rented a room at the Budget Inn Motel on South Tacoma Way.

16. When we went into the room we began using methamphetamine and ecstasy. I smoked and ingested approximately 2-3 grams of methamphetamine and 1 tablet of ecstasy. As a result of the drugs and my lack of sleep, I was out of my mind, unable to think straight, and out of control.

17. Approximately 45 minutes later our argument escalated into a physical altercation and the events that I testified to at trial.

18. After my arrest I was assigned Raymond Thoenig as counsel.

19. Mr. Thoenig and I talked about a diminished capacity defense based upon my extreme intoxication.

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22. John Cain and I talked about a defense of diminished capacity and the drug use and blackout episodes.

23. Mr. Cain suffered from an aneurism and could not continue as my attorney.

24. On October 4th, 2005 was appointed Robert M. Quillian as my counsel.

25. Mr. Quillian was my defense counsel at trial.

26. During my meetings with Mr. Quillian we discussed the fact that at the time of the crimes I was using extremely large amounts of Methamphetamine, and alcohol and that I had taken some Ecstasy. He told me he was going to introduce these facts into the trial and that it would be the primary defense.

27. Mr. Quillian asked for a continuance on February 22nd 2006, to do investigation work and for trial preparation. Mr. Quillian did not dicuss any investigation work with me and I am not aware of any investigations conducted.

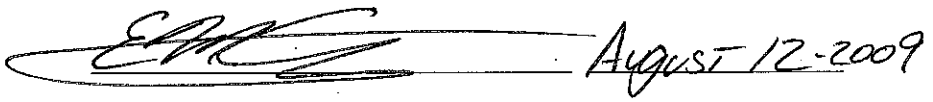
28. I was never presented with any information regarding how the drugs and alcohol use affected my state of mind and we never discussed it further after deciding that we would use it in the defense.

29. Despite repeated attempts to reach him, Mr. Quillian and I had very limited contact prior to the trial.

30. During the pre-trial preparation investigator Becky Durkee came to see me and provided me with a Supplemental Report written by Pierce County Sherriff's Deputy Meyer describing the fact that Officer Henson repeatedly stomped on my head with his boot while I was being held down. I had no recollection of that event.

I declare under penalty of perjury pursuant to the laws of the State of Washington that the foregoing is true and correct to the best of my knowledge.

I declare under penalty of perjury pursuant to the laws of the State of Washington that the foregoing is true and correct to the best of my knowledge.



Edward Michael Glasmann Dated August 12th 2009

Appendix B

Lakewood Police Departm~ Incident No. 042970053.3

Original Supp

Supplemental Report

Quick Print

PDA:	Homeland Security:	Subject:	Aggravated Assault; Strong Arm Robbery; Felony Warrant; Unlawful Imprisonment

Incident No. 042970053.3

IBR Disposition:	Arrest	Case Management Disposition:	
Forensics:		Reporting By/Date:	03-037 - Meyer, Joshua 10/23/2004 5:23:00 AM
Case Report Status:	Approved	Reviewed By/Date:	83-021 - Pebley, William 10/23/2004 9:52:27 PM

Related Cases:

Case Report Number	Agency

Non-Electronic Attachments

Attachment Type	Additional Distribution	Count

Location Address:	10006 S Tacoma Wy	Location Name:	AM PM Mini Market
City, State, Zip:	Lakewood, WA 98444	Cross Street:	
Contact Location:		City, State, Zip:	
CB/Grid/RD:	238 - LAKEWOOD	District/Sector:	LD03 - Lakewood (Southgate)
Occurred From:	10/23/2004 12:46:00 AM Saturday	Occurred To:	
Notes:			

Offense Details: 1205 - Robbery - Street - Weapon

Domestic Violence:	No	Child Abuse:	Gang Related:	Juvenile:	
Completed:	Attempted		Crime Against: PR	Hate/Bias:	None (No Bias)
Criminal Activity:				Using:	
Location Type:	Convenience Store (7-11)		Type of Security:	Tools:	
Total No. of Units Entered:			Evidence Collected:		
Entry Method:					
Notes:					

Offense Details: 1305 - Assault - Aggravated - NonFamily - Weapon

Domestic Violence:	Yes	Child Abuse:	Gang Related:	Juvenile:	
Completed:	Completed		Crime Against: PE	Hate/Bias:	None (No Bias)
Criminal Activity:				Using:	
Location Type:	Parking Lot		Type of Security:	Tools:	
Total No. of Units Entered:			Evidence Collected:		

Call Source:	Field	Assisted By:	03-028 - Sutherland, David
Phone Report:		Notified:	
Insurance Letter:		Entered By:	03-028 - Sutherland, David
Entered On:	10/23/2004 5:23:27 AM	Approved By:	201034 - Hemion, Keri
Approved On:	10/24/2004 1:45:32 AM	Exceptional Clearance:	
Adult/ Juvenile Clearance:		Exceptional Clearance Date:	
Additional Distribution:		Other Distribution:	

Validation Processing	Distribution Date: 10/24 By: JM	County Pros. Atty. X	Juvenile	Other	CPS	Supervisor:
	Indexed Date: By:	City Pros. Atty.	Military	DSHS	PreTrial	

For Law Enforcement Use Only -- No Secondary Dissemination Allowed

Records has the authority to ensure correct agency, CB/Grid/RD, and District/Sector are incorporated in the report.

Printed: October 24, 2004 - 1:45 AM

Printed By: Hemion, Keri

(29)

Entry Method:
Notes:

Offense Details: 1007 - Kidnap Hostage For Escape

Domestic Violence:	Yes	Child Abuse:	Gang Related:	Juvenile:	
Completed:	Completed		Crime Against: PE	Hate/Bias:	None (No Bias)
Criminal Activity:				Using:	
Location Type:	Convenience Store (7-11)		Type of Security:	Tools:	
Total No. of Units Entered:			Evidence Collected:		
Entry Method:					
Notes:					

Offense Details: 4802 - Obstruct Criminal Investigation/Material Witness

Domestic Violence:	No	Child Abuse:	Gang Related:	Juvenile:	
Completed:	Completed		Crime Against:	Hate/Bias:	None (No Bias)
Criminal Activity:				Using:	
Location Type:	Street/Right of Way		Type of Security:	Tools:	
Total No. of Units Entered:			Evidence Collected:		
Entry Method:					
Notes:					

Offense Details: 2203 - Burglary - Forced Entry - Non Residence

Domestic Violence:	No	Child Abuse:	Gang Related:	Juvenile:	
Completed:	Completed		Crime Against: PR	Hate/Bias:	None (No Bias)
Criminal Activity:				Using:	
Location Type:	Convenience Store (7-11)		Type of Security: None	Tools:	Other None
Total No. of Units Entered:			Evidence Collected:		
Entry Method:	Other Unique				
Notes:					

Offense Details: 8910 - Criminal Arrest Warrant

Domestic Violence:	No	Child Abuse:	Gang Related:	Juvenile:	
Completed:	Completed		Crime Against:	Hate/Bias:	None (No Bias)
Criminal Activity:				Using:	
Location Type:	Convenience Store (7-11)		Type of Security:	Tools:	
Total No. of Units Entered:			Evidence Collected:		
Entry Method:					
Notes:					

Weapon 1: Motor Vehicle

Offense:	1305 - Assault - Aggravated - NonFamily - Weapon	Serial No:	
Offender:		OAN:	
Weapon:	Motor Vehicle	Automatic:	
Other Weapon:		Caliber:	

Action:		Gauge:	
Manufacturer:		Length:	
Make:		Finish:	
Importer:		Grips:	
Model:		Stock:	
Weapon Notes:			

Weapon 2: Implied Weapon

Offense:	1205 - Robbery - Street - Weapon	Serial No:	
Offender:		OAN:	
Weapon:	Implied Weapon	Automatic:	
Other Weapon:		Caliber:	
Action:		Gauge:	
Manufacturer:		Length:	
Make:		Finish:	
Importer:		Grips:	
Model:		Stock:	
Weapon Notes:			

Weapon 3: Implied Weapon

Offense:	1007 - Kidnap Hostage For Escape	Serial No:	
Offender:		OAN:	
Weapon:	Implied Weapon	Automatic:	
Other Weapon:		Caliber:	
Action:		Gauge:	
Manufacturer:		Length:	
Make:		Finish:	
Importer:		Grips:	
Model:		Stock:	
Weapon Notes:			

Investigative Information

Means:		Motive:	
Vehicle Activity:		Direction Vehicle Traveling:	

Synopsis:

Narrative: On 10/23/04 at approximately 0049 hours, Deputy Sutherland and I assisted Lakewood Police Officers, regarding a Domestic Violence Kidnapping call at the AMPM 10006 South Tacoma Wy .

Upon our arrival I saw several Lakewood officers at the front entrance trying to negotiate with the suspect inside. The suspect had taken two hostages, the store clerk and a female he might have ran over with his car moments earlier. I ran around the northwest corner of the building in order to secure any rear exits. When other Deputies arrived I continued around to the south east corner to secure another door.

When I came around the corner towards the front of the store, I heard several officers out front saying officers inside were fighting with the suspect in order to place him into custody. Since I didn't know how many officers were inside I ran in to see if I could assist in any way. When I entered the store I saw the cashier's counter, which is "U" shaped with the entrance on the west side of the store. Inside the counter several officers were actively fighting with the suspect. Several other officers were trying to assist by climbing over the counter. As I ran around the counter I saw Sgt. Eakes (LK-16) trying to pull the victim out of the melee`. I started to get a clearer picture of the fight and could see a Lakewood Officer (later identified as Officer Henson LK-122)

standing near the suspect's head. Holding his rifle in his right hand Henson began stomping downward (with his right foot, by raising it approximately 1 to 2 feet off the ground) onto the suspect's head, which was between another officer's feet. Henson stomped once or twice before I grabbed his rifle out of his hands. I saw blood start to appear on the suspect's face. Sgt. Eakes must have also seen what was going on, because he began telling Henson to stop by saying "whoa whoa whoa, knock it off and that's enough", he reached out and touched Henson's left arm while he was yelling. Sgt. Eakes managed to catch Henson's actions even though he was concentrating on removing the victim and getting her to safety.

Henson stopped momentarily and it appeared as if he looked in Sgt. Eakes direction, before he resumed stomping on the top of the suspect's head three more times. I was repeating what Sgt. Eakes had been saying, "Whoa, whoa, whoa!" and when it didn't appear that Henson could hear us I grabbed him by the back of his duty belt and pulled him away from the fight.

Sgt. Eakes managed to pull the victim loose and I escorted her outside to a waiting patrol car to wait for Paramedics. I took the victim's information and then returned Henson's rifle to him after the suspect was removed from the building in handcuffs. The suspect continued to fight with Officer's even when he was handcuffed and finally had to be hog-tied for his and officer's safety.

My supervisor Sgt. Pebley (73) approached me at the scene and asked what I had seen. I relayed to him what I saw and he instructed me to write a Supplemental report.

Later in my shift I spoke to Sgt. Eakes and told him what I saw and that I was writing a Supplemental report as well. He told me that was fine and that he had spoken to Henson about the incident and was notifying his administration of the incident as well. Sgt. Eakes told me he had seen Henson stomp on the suspect at least three times himself.

The entire incident should have been videotaped by the store's security cameras.

-Nothing Further-
Joshua D Meyer
PCSD 494/03037

Reviewed By: Reviewed Date:

Cad Details

Return to Results Screen

Complaint: **20042970053** Disp: E
Address: 9915 S TACOMA WAY
Name: BUDGET INN SOUTH TACOMA
Phone: 2535886616

Call Received: 20041023 0046
Call Cleared: 20041023 0220
End Priority:

	Date/Time	Unit		ID	Station
Dispatch:	20041023 0047	LK131	Dispatcher:	E07899	SD01
Arrival:	20041023 0051	LK131	Com Officer:	E79293	CM07
Clear:	20041023 0220	LK122	Assigned Detective :		
Close:	20041023	LK152			

	Agency	CB	District	Incident Type	Location
Starting:	SW	238	LD03	DV - Domestic	9915 S TACOMA WY
Ending:	SW	238	LD03	DV - Domestic	9915 S TACOMA WY

System Date	System Time	Com	Station	Off	Text
20041023	00:46:36	E911	CM07	E79293	LOCATION:9915 S TACOMA WAY \PHONE:253/588-6616 \COMP:BUDGET INN SOUTH TACOMA
20041023	00:46:36	ENTRY	CM07	E79293	TEXT:AID ADV..MALE HAS RUN OVER A FEMALE IN PARKING LOT, POSS DV \SOURCE:BUSN
20041023	00:46:36	PRIOR	CM07	E79293	LP MVT 10/21/04 @ 09:18:27 (50 MORE)
20041023	00:47:33	DISPATCH	SD01	E07899	LK131
20041023	00:47:33	ID	SD01	E07899	LK131 <L04021>HAMILTON, RYAN <L04009>HECTOR, MICHELLE
20041023	00:48:00	SUPP	CM07	E79293	TEXT:1 AGO, SB ON STW, BLK CORVETTE POSS LIC 354PVD (WA)...FEMALES LEGS ARE HANGING OUT OF THE CAR..
20041023	00:48:35	BACKUP	SD01	E07899	LK131 LK16
20041023	00:48:35	ID	SD01	E07899	LK16 <L04002>EAKES, MARK
20041023	00:49:04	SUPP	CM07	E79293	TEXT:SUSPECT IS W/M,35YR,6'0,THIN,LG BLO,BLK LEATHER JACKET,RED T SHIRT,JEANS,..HE BEAT UP THE FEMALE, AND THEN RAN OVER HER AND THEN KEPT THE TIRE ON THEN SPUN HIS TIRES ON TOP OF HER, RAN HER OVER SEVARAL TIMES
20041023	00:49:12	BACKUP	SD02	E04390	LK16 488
20041023	00:49:12	ID	SD02	E04390	488 <C03026>DAVIDSON, PATRICK A
20041023	00:49:13	CHGLOCOS	SD01	E07899	LK16 100TH ST SW/S TACOMA WY ,LD, AMPM , CARJACKING IN PROGRESS
20041023	00:49:26	MISC	SD03	E06699	LK16, FIGTHING W/ONE
20041023	00:49:29	MISC	SD03	E06699	#53, WSP ADV
20041023	00:49:29	MISC	SD01	E07899	LK16, GUNPT
20041023	00:49:33	BACKUP	SD03	E06699	LK16 490
20041023	00:49:33	ID	SD03	E06699	490 <C03028>SUTHERLAND, DAVID <C03037>MEYER, JOSHUA
20041023	00:49:50	BACK-OS	SD01	E07899	LK16 LK152
20041023	00:49:50	ID	SD01	E07899	LK152 <L04006>BORCHARDT, TIMOTHY <L04019>BUTTS, DAVID
20041023	00:49:55	BACKUP	PD02	E06593	LK152 U31 U47 J51 U33

20041023	00:49:55	ID	PD02	E06593	U31 <T35760>HENSLEY, DANIEL L
20041023	00:49:55	ID	PD02	E06593	U47 <T09431>LANE, RYAN
20041023	00:49:55	ID	PD02	E06593	J51 <T12139>GETTS, HENRY <T12140>FRISBIE, RANDALL
20041023	00:49:55	ID	PD02	E06593	U33 <T03203>ONEILL, PATRICK T
20041023	00:50:01	BACKUP	PD02	E06593	U31 H36
20041023	00:50:01	ID	PD02	E06593	H36 <T52903>LOYD, GARY H <T07981>BAMBICO, DANILO R
20041023	00:50:06	SUPP	CM07	E79293	TEXT:HE THAN GRADDED HER AND THREW HER IN THE CAR WITH LEGGS HANGING OUT...RP IS ERICA RUSK, HME 253 536 7103...REFUSED TO GIVE ROOM # SHE IS STAYING IN
20041023	00:50:10	MISC	SD01	E07899	490, HE HAS A HOSTAGE
20041023	00:50:35	MISC	SD01	E07899	LK16, CALL OUT SWAT AND CDO
20041023	00:50:37	BACKUP	SD02	E04390	488 235
20041023	00:50:37	ID	SD02	E04390	235 <C01004>CONNER, JASON R <C84016>TIFFANY, GERALD D
20041023	00:50:44	BACKUP	SD01	E07899	LK16 415
20041023	00:50:44	ID	SD01	E07899	415 <C00030>CAREY, MARIO D
20041023	00:50:44	MISC	SD03	E06699	LK16, CALLING SUMNER FOR SWAT
20041023	00:50:45	BACK-ER	PF35	C00053	LK16 430
20041023	00:50:45	ID	PF35	C00053	430 <C00053>PHIPPS, MICHAEL <C00020>ONEIL, ANDREW J
20041023	00:50:49	BACKUP	SD02	E04390	488 313
20041023	00:50:49	ID	SD02	E04390	313 <C94038>SHEARS, CORY W <C95011>BERRY, MARK C
20041023	00:51:15	MISC	SD02	E04390	313, DROPPING OFF PARTNER 321 TO ASSIST AND GET HIS GEAR FOR SWAT
20041023	00:51:18	ONSCENE	PD02	E06593	J51
20041023	00:52:03	MISC	SD03	E06699	LK16, SUMNER ADV TO PAGE SWAT OUT TO RESPOND TO LOCATION TIL WE FIND OUT WHERE THEY WANT THEM TO STAGE
20041023	00:52:13	MISC	SD01	E07899	LK16, OIC
20041023	00:52:24	MISC	SD01	E07899	LK16, CANCEL SWAT
20041023	00:52:31	INSRVICE	PF35	C00053	430
20041023	00:52:32	ONSCENE	PD02	E06593	U31
20041023	00:52:43	INSRVICE	PD02	E06593	H36
20041023	00:52:50	BACK-OS	SD01	E07899	LK16 424, AID PRIORITY FOR THE SUSP
20041023	00:52:50	ID	SD01	E07899	424 <C00045>RICKERSON, MARK A
20041023	00:52:57	INSRVICE	SD02	E04390	235
20041023	00:52:58	BACK-OS	SD01	E07899	LK16 SM12
20041023	00:52:58	ID	SD01	E07899	SM12 <SM2535>RETTIG, MARC
20041023	00:52:59	INSRVICE	SD02	E04390	313
20041023	00:53:07	PREEMPT	PD02	E06593	U33
20041023	00:53:28	BACKUP	PD02	E06593	U31 U38
20041023	00:53:28	ID	PD02	E06593	U38 <20035#>BORGES, THOMAS

CAD Details

20041023	00:53:33	PREEMPT	PD02	E06593	U38
20041023	00:53:35	INSRVICE	PD02	E06593	U47
20041023	00:53:40	MISC	SD03	E06699	LK16, SUMNER ADV TO CANCEL SWAT..
20041023	00:53:43	MISC	SD03	E06699	LK16, LK1 ADV
20041023	00:54:04	MISC	SD01	E07899	LK152, AID ROUTINE
20041023	00:54:09	MISC	SD01	E07899	LK152, CANCEL DEEPER
20041023	00:54:54	ONSCENE	PC76	C03026	488
20041023	00:57:37	MISC	SD01	E07899	LK16, NEED THE CDO AND FARRAR TO CALL ME ON MY CELL
20041023	00:58:28	MISC	SD01	E07899	LK16, PRETTY SURE THE FEMALE HE TOOK HOSTAGE WAS THE ONE THAT HAD HER LEGS HANGING OUT OF THE VEH , SHE FLED THE VEH AND RAN INTO THE AMPM AND HID BEHIND THE COUNTER WHERE HE LANDED ON TOP OF HER
20041023	00:59:33	BACK-OS	SD01	E07899	LK152 LK122
20041023	00:59:33	ID	SD01	E07899	LK122 <L04052>HENSON, KENNETH <L04073>VAHLE, JEREMY
20041023	00:59:40	MISC	SD01	E07899	LK122, GLASMANN, ED M 102264
20041023	01:01:09	INSRVICE	PF37	C00045	424
20041023	01:01:18	PREEMPT	PD02	E06593	J51
20041023	01:02:01	RCONTACT	SD01	E07899	LK152
20041023	01:02:07	MISC	SD03	E06699	LK16, FARRAR ADV
20041023	01:02:51	MISC	LA02	L04006	LK152, STATE MESSAGE FOR LAW ENFORCEMENT SUPPORT AGENCY TM273.L..WA0272373.LIC/544PZO VIN/1G1YY0788G5106712 .YYR/1986.VMA/CHEV .VMO/CVTCP EXP DATE/05-06-2005 GRIMES,ROBERT DEAN PIC NAME1 GRIMERD404OA 8530 STEILACOOM RD SE UNIT 35 TAD# IS RB75341 05 OLYMPIA,WA,98513 PREV TAB F623629 04 LEGAL OWNER SAME AS ABOVE PLATE ISSUE DATE/ 05-2003 TITL
20041023	01:05:09	INSRVICE	SM49	SM2535	SM12
20041023	01:05:15	RCONTACT	SD01	E07899	U31
20041023	01:06:00	INSRVICE	PC76	C03026	488
20041023	01:07:47	MISC	SD01	E07899	LK152, TOW
20041023	01:09:08	MISC	SD03	E06699	LK152, TACOMA 15
20041023	01:10:53	RCONTACT	SD01	E07899	LK122
20041023	01:11:07	INSRVICE	TC15	T35760	U31
20041023	01:11:39	BACK-OS	SD01	E07899	LK122 LK153
20041023	01:11:39	ID	SD01	E07899	LK153 <L04007>LOFLAND, JIM <L04014>PORCHE, CHARLES
20041023	01:25:40	RCONTACT	SD01	E07899	LK153
20041023	01:29:30	ONSCENE	PC81	C03028	490
20041023	01:34:04	INSRVICE	PF74	C00030	415
20041023	01:38:01	PREEMPT	SD01	E07899	490
20041023	01:40:35	TRANSPRT	SD01	E07899	LK122 JAIL, W/1 MALE
	01:50:18				

CAD Details

20041023		PREEMPT	SD01	E07899	LK16
20041023	01:51:56	PREEMPT	SD01	E07899	LK152
20041023	01:53:00	PREEMPT	SD01	E07899	LK131
20041023	02:00:29	MISC	SD01	E07899	.59, 490 CLEAR W/SUPP
20041023	02:20:19	TR-CMPT	SD01	E07899	LK122
20041023	02:20:37	CLEAR	SD01	E07899	LK122 F
20041023	02:21:56	BACKUP	SD01	E07899	LK153 LK152
20041023	02:21:59	ID	SD01	E07899	LK152 <L04006>BORCHARDT, TIMOTHY <L04019>BUTTS, DAVID
20041023	02:22:03	CHGLOC	SD01	E07899	LK152 TG
20041023	02:25:14	INSRVICE	LA06	L04007	LK153
20041023	02:25:50	DISPOSTN	SD01	E07899	E
20041023	02:36:32	MISC	SD03	E06699	LK152, DOING A CT SCAN ON HER NOW...MORE THAN LIKELY WILL BE RELEASED TONIGHT..WILL NO MORE AFTER CT SCAN
20041023	03:13:02	ONSCENE	SD03	E06699	LK152
20041023	03:24:51	RCONTACT	SD01	E07899	LK152
20041023	03:41:32	CLEAR	LA02	L04006	LK152 E, ATTEMPTED ROBBERY 1 X 3 ASLT 1 DV UNLAWFUL IMPRISONMENT OBSTRUCTING WRT
20041023	03:41:32	CLOSE	LA02	L04006	LK152 E, ATTEMPTED ROBBERY 1 X 3 ASLT 1 DV UNLAWFUL IMPRISONMENT OBSTRUCTING WRT

Appendix C

IN THE UNITED STATES DISTRICT COURT

WESTERN DISTRICT OF WASHINGTON AT TACOMA

EDWARD M. GLASMANN,)

Plaintiff,)

vs.) NO. C07-5193-RBL

PIERCE COUNTY SHERIFF'S OFFICE;)

PIERCE COUNTY, WASHINGTON;)

LAKEWOOD POLICE DEPARTMENT;)

CITY OF LAKEWOOD, WASHINGTON,)

Defendants.)

DEPOSITION UPON ORAL EXAMINATION

OF

ANGEL MARIE BENSON

1:28 p.m.

October 13, 2008

6000 Main Street Southwest, Conference Room 3C

Lakewood, Washington

Brenda Steinman

Court Reporter

A. Yeah.
Q. Or Michael is his middle name?
A. Yeah.
Q. So at some point while Mike was in that work release program you two began dating, is that correct?
A. Yes.
Q. At some point during that dating did he move in with you? Did you move in together?
A. Yes, he released from work release to my address.
Q. What address was that?
A. Lebanon Street, I want to say 1815 -- no, that was or Highland Drive. I don't remember.
Q. But it was an address on Lebanon Street?
A. 1317, that's it.
Q. 1317?
A. Lebanon Street.
Q. And that's in what city?
A. Lacey.
Q. Can you describe for me -- this incident that is the subject of this lawsuit, like I said, occurred sometime in approximately October 2004. You were in a relationship with Mr. Glasmann in the months leading up to that, is that correct?
A. Yes.

Q. Can you describe for me kind of how the relationship was during that time period.
A. The whole time?
Q. Well, maybe more at the beginning.
A. It was good at the beginning. He was clean. And he -- when he released to my address he then was -- he found a three bedroom mobile home with a shop that he was purchasing owner, lease/owner, or owner/contract, whatever it was. And he was doing mechanic work on the side, and he was working for DK Concrete, and, you know, things seemed to be going pretty well.
Then it was like a month, I want to say like a month before what happened with this happened things changed fast.
Q. Why do you think things changed, do you know?
A. He started using.
Q. He started using?
A. Meth.
Q. Meth?
A. Uh-huh.
Q. Did both of you start using meth?
A. Not together.
Q. But at some point Mike started using meth,

is that correct?
A. Yes.
Q. Did you notice any changes in him after he started using meth?
A. Yes, he was -- even his mannerisms changed, his eyes changed, his voice changed. It was like two different people.
Q. When you say "changed," can you describe for me kind of what you mean by that?
A. Like Jekyll and Hyde kind of thing.
Q. I take it you liked him when he wasn't using meth.
A. He was a totally different person, totally.
Q. Can you describe for me kind of what was different?
A. He would do things like I would leave the car unlocked and he would steal the stereo system out of it. Then I would -- he would be really, really mad at me because the stereo system got stolen, and then I would find the stereo system. Like I sat down on the bed in the extra bedroom and it was hidden under the blankets. You know, just weird, weird stuff like that.
Q. I'm assuming that you were in a relationship with him earlier because you liked him, is that

correct?
A. Yeah, he was a nice guy.
Q. Did he seem like a nice guy while he was using meth?
A. No, not on that he wasn't. And he probably wasn't a nice guy when he wasn't. You know, my man picker is broke.
Q. Your what?
A. My man picker is broke.
Q. Meaning your choice of --
A. My choice of nice guys are usually not nice.
Q. When he was using meth you indicated he wasn't a nice guy. Was he violent or mean towards you at all during that time?
A. He was only violent with me one other time before the night that all this stuff happened up here. I called him a punk and I was across the room from him and he threw a shoe at me and hit me in the back with it. He came raging up on me and started punching and punching and punching and would not stop punching. That was the only other time. That came completely out of nowhere. That's part of the Jekyll and Hyde, how he changed.
Q. How long ago was that before the incident roughly, do you know?

Appendix D

Washington State Court of Appeals
Division Two

950 Broadway, Suite 300, Tacoma, Washington 98402-4454
David Ponzoha, Clerk/Administrator (253) 593-2970 (253) 593-2806 (Fax)
General Orders, Calendar Dates, Issue Summaries, and General Information at http://www.courts.wa.gov/courts

January 23, 2008

Kathleen Proctor
Pierce County Prosecuting Atty Ofc
930 Tacoma Ave S Rm 946
Tacoma, WA, 98402-2171

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4616 25th Ave NE # 552
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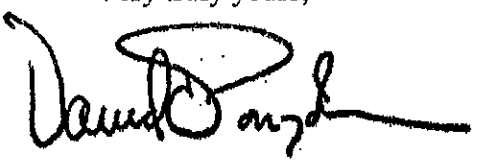
Edward Michael Glassman
#905293
Monroe Corr Complex
P.O. Box 777
Monroe WA 98272-0777

CASE #: 34997-3-II
State of Washington, Respondent v Edward M. Glassman, Appellant

Counsel:

An opinion was filed by the court today in the above case. A copy of the opinion is enclosed.

Very truly yours,



David C. Ponzoha
Court Clerk

DCP:cjb
Enclosure

cc: Judge Beverly Grant
Indeterminate Sentence Review Board

FILED
COURT OF APPEALS
DIVISION II
08 JAN 23 AM 9:14
STATE OF WASHINGTON
BY
DEPUTY

IN THE COURT OF APPEALS OF THE STATE OF WASHINGTON

DIVISION II

STATE OF WASHINGTON, Respondent, v. EDWARD MICHAEL GLASMANN, Appellant.	No. 34997-3-II UNPUBLISHED OPINION

HUNT, J. — Edward Michael Glasmann appeals his jury conviction for second degree assault.[1] He argues the State failed to establish that he intentionally ran over the victim's leg with his car. In his statement of additional grounds (SAG),[2] he asserts that (1) he was denied his right to a fair trial because members of the jury allegedly observed him in handcuffs, and (2) he was denied effective assistance of counsel because his attorney failed to request a voluntary intoxication instruction. We affirm.

FACTS

I. ASSAULT

Edward Michael Glasmann and Angel Benson were romantically involved and engaged to be married. On the night of October 22, 2005, Glasmann and Benson went to dinner in Tacoma and rented a motel room in Lakewood to celebrate Glasmann's birthday. Both

[1] The jury also convicted Glasmann of attempted second degree robbery, first degree kidnapping, and obstructing a law enforcement officer. He does not challenge those convictions in this appeal.

Glasmann and Benson ingested methamphetamine, ecstasy, and alcohol over the course of the evening. In addition, Glasmann and Benson had been arguing throughout that day and evening.

Around midnight, their argument escalated. Glasmann hit Benson, who curled up into the fetal position to protect herself from his blows. Glasmann eventually told Benson that he wanted to go for a ride. They both left the motel room.

Outside the room, another hotel guest, Erika Rusk, witnessed Glasmann (1) pin Benson against the wall with one hand around her neck and repeatedly punch her with his other hand; (2) release Benson and kick her twice in the stomach; (3) drag her to the passenger side of his Corvette and got into the driver's seat; (4) reach over to the open passenger door and attempt to pull Benson into the car by her hair; (5) pull forward from the parking stall while Benson was not fully in the car; and (6) run over Benson's leg with his car.

Once in the car, Benson put the car into park, grabbed the keys, and ran into a mini-mart adjacent to the motel. Inside the mini-mart, she hid on the floor behind the counter. As Rusk watched, she was calling 911 and reporting these events to dispatch.

Lakewood Police Officers Timothy Borchardt and David Butts arrived to find Glasmann's Corvette parked in the roadway. As they approached, they observed Glasmann exit his Corvette, run over to the mini-mart, and climb into three separate cars, apparently hoping to steal one and escape.

Their guns drawn, Officers Borchardt and Butts ordered Glasmann to show his hands. Glasmann refused to comply, and told the officers that he had a gun. When Glasmann pushed a

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[2] RAP 10.10.

man aside in order to access the third car, Officer Butts approached the open driver's side window and sprayed pepper spray into Glasmann's eyes. Glasmann then exited the vehicle through the passenger door and ran into the mini-market, pursued by a group of officers.

Glasmann continued to yell, "[S]hoot me, I have got a gun. Go ahead and shoot me." 4 Report of Proceedings (RP) at 116. As if it were a weapon, he pointed a black object at the officers. Eventually, Glasmann ran behind the counter, grabbed Benson, put his arm around her neck in a choke hold, and pulled her body in front of his, threatening to kill her. Glasmann then dropped to the floor, holding Benson between him and the officers.

When Benson was able to "wiggle her way down from [Glasmann's] body," Officer Ryan Hamilton applied a stun gun to Glasmann. 4 RP at 125-26. The officers then removed Benson. They took Glasmann into custody, determined he was not armed, and realized he had brandished a stereo remote control as a weapon.

II. FOLLOW-UP

Benson was taken to Tacoma General Hospital, where Dr. William Eggebroten examined and treated her injuries: several contusions and abrasions on her right leg, hip, and arms. While at the hospital, Officer Borchardt and Officer Butts interviewed Benson about the incident. She told them that Glasmann had threatened to kill her if she did not get into his Corvette in the motel parking lot. Benson was released a few hours after arriving at the hospital.

A few days later, on October 27, 2004, the Lakewood police domestic violence detective met with Benson to conduct a follow-up interview. The detective examined only those injuries that Benson's clothing did not cover. He did not take pictures at the time because they were in a

public place. But Benson agreed to have a friend take pictures of her injuries and send them to him the following morning.

III. PROCEDURE

The State charged Glasmann with one count of first degree assault under RCW 9A.36.011(1)(a); one count of attempted first degree robbery under RCW 9A.56.190, .200; RCW 9A.28.020; one count of first degree kidnapping under RCW 9A.40.020(1)(a); and one count of obstructing a law enforcement officer under RCW 9A.76.020(1).

At trial, Glasmann, Benson, the officers, and Rusk all testified, resulting in conflicting testimony as to the events that occurred on the night in question. The State also submitted the 911 dispatch tape,[3] the mini-mart surveillance tape,[4] and the recorded conversations between Glasmann and Benson while Glasmann was in the Pierce County Jail awaiting trial.

Apparently on one occasion, three jurors observed Glasmann in handcuffs outside the courtroom. Glasmann's counsel requested a mistrial. The trial court questioned jurors number three and thirteen about their observations of Glasmann outside the courtroom. Both jurors testified that they did not form impressions of Glasmann based on their observations of him in the hallway. One of the jurors testified that he saw Glasmann on the elevator "for a second." The other juror testified that he turned a corner in the courthouse and saw Glasmann for "a split

[3] On the 911 tape, Rusk describes the events as they occur: Glasmann pulled forward in his vehicle, backed up, and then pulled forward again, driving over Benson's leg three times. Glasmann then reached over, yanked Benson into the car, and pulled out of the parking lot, onto South Tacoma Way.

[4] The surveillance video showed the events inside the mini-mart.

second" before he turned around and left. When the court asked the two jurors whether they could follow an instruction telling them that they were not to consider the fact that they had seen Glasmann in the hall, both answered, "Yes."

The officer who had been transporting Glasmann testified that the jurors who observed Glassman were between eight and ten feet away, and he (the officer) did not believe the jurors saw the handcuffs, because Glasmann was wearing a long sleeved shirt that covered the handcuffs and was holding a book in his hands. The trial court found no prejudicial effect and denied Glasmann's motion for mistrial.

The jury convicted Glasmann of second degree assault, attempted second degree robbery, first degree kidnapping, and obstructing a law enforcement officer. Glasmann stipulated to his offender score, and the trial court sentenced him to a standard range sentence, totaling 198 months.

Glasmann appeals.

ANALYSIS

I. SUFFICIENCY OF EVIDENCE

Glasmann argues that the State failed to present sufficient evidence to prove the requisite intent to convict him of second degree assault. His argument fails.

A. Standard of Review

Sufficiency of the evidence is a question of constitutional magnitude, which an appellant may raise for the first time on appeal. *State v. Alvarez*, 128 Wn.2d 1, 13, 904 P.2d 754 (1995). When a defendant challenges the sufficiency of the evidence in a criminal case on appeal, we

draw all reasonable inferences from the evidence in favor of the State and interpret all reasonable inferences from the evidence strongly against the defendant. *State v. Partin*, 88 Wn.2d 899, 906-07, 567 P.2d 1136 (1977). A claim of sufficiency admits the truth of the State's evidence and all inferences that an appellate court can reasonably draw therefrom. *State v. Theroff*, 25 Wn. App. 590, 593, 608 P.2d 1254, *aff'd*, 95 Wn.2d 385 (1980).

Evidence is sufficient to support a conviction if, after viewing the evidence in the light most favorable to the State, any rational trier of fact would have found guilt beyond a reasonable doubt for the crime charged. *State v. Green*, 94 Wn.2d 216, 221-22, 616 P.2d 628 (1980). We must defer to the trier of fact on issues of conflicting testimony, credibility of witnesses, and the persuasiveness of the evidence. *State v. Thomas*, 150 Wn.2d 821, 874-75, 83 P.3d 970 (2004).

B. Second Degree Assault

RCW 9A.36.021(1)(a) provides: "A person is guilty of assault in the second degree if he or she, under circumstances not amounting to assault in the first degree: . . . Intentionally assaults another and thereby recklessly inflicts substantial bodily harm."

> Recklessly causing harm is not the same as intentionally causing harm. Thus, under the statute, second degree assault by battery requires an intentional touching that *recklessly* inflicts substantial bodily harm. It does not require specific intent to inflict substantial bodily harm.

State v. Esters, 84 Wn. App. 180, 185, 927 P.2d 1140 (1996), *review denied*, 131 Wn.2d 1024 (1997); *see also State v. Keend*, 140 Wn. App. 858, ___, 166 P.3d 1268, 1273 (2007); *State v. Walden*, 67 Wn. App. 891, 893-94, 841 P.2d 81 (1992) (defendant may be convicted of second

degree assault if he intended to put another in apprehension of harm whether or not he intended to inflict or was incapable of inflicting that harm).

The State presented sufficient evidence to establish that Glasmann intentionally touched Benson, thereby recklessly inflicting substantial bodily harm. Rusk testified about the events she had observed from outside her motel room: Glasmann dragged Benson into the passenger seat of his car, got into the driver's side, and drove forward while Benson was only half in the vehicle. Glasmann drove over Benson's leg, reversed the car, and then pulled forward again onto her leg. And after running over her leg three times, Glasmann yanked Benson into the car by her hair and drove off. The State also presented the 911 dispatch tape, which corroborated Rusk's testimony.

Although each provided slightly different details about the events, Benson,[5] the officers, and the physician who had examined Benson after the assaults, all testified. The jury also heard recorded telephone calls that Glasmann had made to Benson while awaiting trial, suggesting that they discussed details of their testimony before trial and/or that he threatened Benson to testify in a specific way at trial.

[5] Benson testified that she and Glasmann had been engaged in an ongoing verbal argument throughout the day, which escalated into a mutual physical altercation later at the motel. The altercation moved from inside their motel room to outside by the car because Glasmann wanted to go for a drive. Although Benson did not want to go with Glasmann because she was afraid of him driving, somehow she ended up in the passenger seat. She opened the door and tried to get out while the car was moving. While she held onto something or Glasmann held onto her from the driver's seat, Benson was running backwards, trying to catch her balance; she fell, and the car went up her leg and parked on her pelvis. Glasmann then reversed the car off Benson, got out, put her back in the passenger seat, and said he was taking her to the hospital. But Benson was scared, so she put the car in park, grabbed the keys, and ran to the mini-mart. Inside the mini-mart, Benson yelled, "Help me," or "Save me," and hid behind the counter.

Glasmann testified that he had intentionally pushed Benson into the car, even though she had made it clear that she did not want to drive with him. He even acknowledged that his car had "rolled" onto Benson, after which he yanked her into the car. He claimed, however, that he had driven out of the motel parking lot with the intention of finding a hospital for her.

In essence, the jury had to weigh the conflicting testimonies of Glasmann, Benson, and the other witnesses. We defer to the jury's finding Glasmann and Benson not credible.[6] Taken in the light most favorable to the State, the testimony provided sufficient evidence for the jury to find that Glasmann intentionally touched Benson when he dragged her into the car, pulled her hair, and ran over her leg, among other intentional touches, thereby recklessly inflicting substantial bodily harm, namely several contusions and abrasions on her right leg, hip, and arms. Accordingly, we hold that the State presented sufficient evidence to prove the elements of second degree assault.

II. Statement of Additional Grounds

A. Right to Fair Trial

In his SAG, Glasmann first contends that the trial court denied his right to a fair trial under the federal and Washington state constitutions because members of the jury observed him in handcuffs outside the courtroom. We disagree.

On appeal, we evaluate an unconstitutional restraint claim under a harmless error standard. *State v. Finch*, 137 Wn.2d 792, 861, 975 P.2d 967, *cert. denied*, 528 U.S. 922 (1999).

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[6] *State v. Cord*, 103 Wn.2d 361, 367, 693 P.2d 81 (1985); *State v. Casbeer*, 48 Wn. App. 539, 542, 740 P.2d 335, *review denied*, 109 Wn.2d 1008 (1987).

We presume an error violating a constitutional right to be prejudicial, unless it affirmatively appears from the record to be harmless beyond a reasonable doubt. *Finch*, 137 Wn.2d 859. Harmless error may be established when the evidence against the defendant is so overwhelming that no rational conclusion other than guilt can be reached. *Finch*, 137 Wn.2d at 859.

But when the jury's view of the defendant in shackles or handcuffs is brief or inadvertent, the defendant must make an affirmative showing of prejudice, and he carries the burden of curing any defect. *State v. Elmore*, 139 Wn.2d 250, 273, 985 P.2d 289 (1999), *cert. denied*, 531 U.S. 837 (2000). To demonstrate prejudice, the defendant must show "'a substantial or injurious effect or influence on the jury's verdict.'" *Elmore*, 139 Wn.2d at 274 (quoting *State v. Hutchinson*, 135 Wn.2d 863, 888, 959 P.2d 1061 (1998)). There must be evidence in the record beyond the defendant's bare allegations that seeing the defendant in shackles prejudiced the jury. *State v. Gosser*, 33 Wn. App. 428, 435, 656 P.2d 514 (1982).

Glasmann fails to persuade us that some jurors observing him in handcuffs outside the courtroom influenced the jury's verdict to his prejudice. *See State v. Damon*, 144 Wn.2d 686, 692, 25 P.3d 418 (2001). The record does support this contention: Neither juror testified that they had observed Glasmann in handcuffs outside the courtroom. Moreover, both jurors testified that they did not form impressions of Glasmann based on their observations of him in the hallway. In addition, the transporting officer explained to the trial court that the jurors had observed Glasmann from between eight and ten feet away and he did not believe they saw the handcuffs, because they were covered by Glasmann's long-sleeved shirt and a book he was holding in his hands. The record shows no prejudice.

We hold, therefore, that the jurors' inadvertent observations of Glasmann outside of the courtroom did not affect his right to a fair trial.

B. Effective Assistance of Counsel

Finally, Glasmann contends that his attorney rendered ineffective assistance of counsel by failing to request an intoxication instruction. This argument also fails.

A criminal defendant is entitled to a voluntary intoxication instruction if: (1) one of the elements of the crime charged is a particular mental state; (2) there is substantial evidence of ingesting an intoxicant; and (3) the defendant presents evidence that this activity affected his ability to acquire the required mental state. *State v. Harris*, 122 Wn. App. 547, 552, 90 P.3d 1133 (2004). In other words, the evidence must reasonably and logically connect Glasmann's intoxication with his asserted inability to form the requisite level of culpability to commit second degree assault. *See State v. Griffin*, 100 Wn.2d 417, 418-19, 670 P.2d 265 (1983); *State v. Kruger*, 116 Wn. App. 685, 692, 67 P.3d 1147 (2003) (stating that mere intoxication is not enough; rather, the evidence must show the effects of the intoxicant).

Glasmann relies on a Division Three case, *State v. Kruger*, to support his contention that he was entitled to a voluntary intoxication jury instruction. In *Kruger*, however, Division Three found "ample evidence of [the defendant's] level of intoxication on both his mind and body, e.g., his 'blackout,' vomiting at the station, slurred speech, and imperviousness to pepper spray." *Kruger*, 116 Wn. App. at 692. But such is not the state of the evidence here.

Contrary to Glasmann's assertion, the record does not contain ample evidence that his level of intoxication affected his ability or lack thereof to form the mental state required to

establish the crimes charged. At best, the evidence merely showed that Glasmann had ingested unspecified amounts of methamphetamine, ecstasy, and alcohol the night of the incident. *See Kruger*, 116 Wn. App. at 692. As such, Glasmann was not entitled to an involuntary intoxication instruction.

Because counsel's performance was not deficient, we hold that Glasmann was not denied effective assistance of counsel when his counsel failed to request an intoxication instruction.

Affirmed.

A majority of the panel having determined that this opinion will not be printed in the Washington Appellate Reports, but will be filed for public record pursuant to RCW 2.06.040, it is so ordered.

Hunt, J.

We concur:

Bridgewater, J.

Penoyar, J.

Appendix E

NIDA InfoFacts: Methamphetamine

PDF Version [272K] Printer-friendly Version En Español

Methamphetamine is a central nervous system stimulant drug that is similar in structure to amphetamine. Due to its high potential for abuse, methamphetamine is classified as a Schedule II drug and is available only through a prescription that cannot be refilled. Although methamphetamine can be prescribed by a doctor, its medical uses are limited, and the doses that are prescribed are much lower than those typically abused. Most of the methamphetamine abused in this country comes from foreign or domestic superlabs, although it can also be made in small, illegal laboratories, where its production endangers the people in the labs, neighbors, and the environment.

How Is Methamphetamine Abused?

Methamphetamine is a white, odorless, bitter-tasting crystalline powder that easily dissolves in water or alcohol and is taken orally, intranasally (snorting the powder), by needle injection, or by smoking.

How Does Methamphetamine Affect the Brain?

Methamphetamine increases the release and blocks the reuptake of the brain chemical (or neurotransmitter) dopamine, leading to high levels of the chemical in the brain, a common mechanism of action for most drugs of abuse. Dopamine is involved in reward, motivation, the experience of pleasure, and motor function. Methamphetamine's ability to rapidly release dopamine in reward regions of the brain produces the intense euphoria, or "rush," that many users feel after snorting, smoking, or injecting the drug.

Chronic methamphetamine abuse significantly changes how the brain functions. Noninvasive human brain imaging studies have shown alterations in the activity of the dopamine system that are associated with reduced motor skills and impaired verbal learning.[1] Recent studies in chronic methamphetamine abusers have also revealed severe structural and functional changes in areas of the brain associated with emotion and memory,[2,3] which may account for many of the emotional and cognitive problems observed in chronic methamphetamine abusers.

Repeated methamphetamine abuse can also lead to addiction—a chronic, relapsing disease, characterized by compulsive drug seeking and use, which is accompanied by chemical and molecular changes in the brain. Some of these changes persist long after

NIDA INFOFA

Recommended Rea

- NIDA Research Repo Methamphetamine: A and Addiction
- Community Drug Ale Bulletin: Methamphetamine

Other NIDA Web S

- NIDA for Teens: Minc Over Matter - Methamphetamine
- NIDA for Teens: Sara Quest - Methamphetamine

methamphetamine abuse is stopped. Reversal of some of the changes, however, may be observed after sustained periods of abstinence (e.g., more than 1 year).[4]

What Other Adverse Effects Does Methamphetamine Have on Health?

Taking even small amounts of methamphetamine can result in many of the same physical effects of other stimulants, such as cocaine or amphetamines, including increased wakefulness, increased physical activity, decreased appetite, increased respiration, rapid heart rate, irregular heartbeat, increased blood pressure, and hyperthermia.

Long-term methamphetamine abuse has many negative health consequences, including extreme weight loss, severe dental problems ("meth mouth"), anxiety, confusion, insomnia, mood disturbances, and violent behavior. Chronic methamphetamine abusers can also display a number of psychotic features, including paranoia, visual and auditory hallucinations, and delusions (for example, the sensation of insects crawling under the skin).

Transmission of HIV and hepatitis B and C can be consequences of methamphetamine abuse. The intoxicating effects of methamphetamine, regardless of how it is taken, can also alter judgment and inhibition and lead people to engage in unsafe behaviors, including risky sexual behavior. Among abusers who inject the drug, HIV and other infectious diseases can be spread through contaminated needles, syringes, and other injection equipment that is used by more than one person. Methamphetamine abuse may also worsen the progression of HIV and its consequences. Studies of methamphetamine abusers who are HIV-positive indicate that HIV causes greater neuronal injury and cognitive impairment for individuals in this group compared with HIV-positive people who do not use the drug.[5,6]

What Treatment Options Exist?

Currently, the most effective treatments for methamphetamine addiction are comprehensive cognitive-behavioral interventions. For example, the Matrix Model—a behavioral treatment approach that combines behavioral therapy, family education, individual counseling, 12-step support, drug testing, and encouragement for non–drug-related activities—has been shown to be effective in reducing methamphetamine abuse.[7] Contingency management interventions, which provide tangible incentives in exchange for engaging in treatment and maintaining abstinence, have also been shown to be effective.[8] There are no medications at this time approved to treat methamphetamine addiction; however, this is an active area of research for NIDA.

How Widespread Is Methamphetamine Abuse?

Monitoring the Future Survey*

According to the 2008 Monitoring the Future survey—a national survey of 8th-, 10th-, and 12th- graders, methamphetamine abuse among students has shown a general decline in recent years; however, it remains a concern. Survey results show that 2.3 percent of 8th-graders, 2.4 percent of 10th-graders, and 2.8 percent of 12th-graders have used methamphetamine in their lifetime. In addition, 0.7 percent of 8th-graders, 0.7 percent of 10th-graders, and 0.6 percent of 12th-graders were current (past-month) methamphetamine abusers. Past-year use of methamphetamine remained steady across all grades surveyed from 2007 to 2008.

Methamphetamine Prevalence of Abuse
Monitoring the Future Survey, 2008

	8th Grade	10th Grade	12th Grade
Lifetime**	2.3%	2.4%	2.8%
Past Year	1.2	1.5	1.2
Past Month	0.7	0.7	0.6

National Survey on Drug Use and Health***
The number of individuals aged 12 years or older reporting past-year methamphetamine use declined from 1.9 million in 2006 to 1.3 million in 2007. An estimated 529,000 Americans were current (past-month) users of methamphetamine (0.2 percent of the population). Of the 157,000 people who used methamphetamine for the first time in 2007, the mean age at first use was 19.1 years, which is down from the mean age of 22.2 in 2006.

Other Information Resources

For more information on the effects of methamphetamine abuse and addiction, visit www.drugabuse.gov/drugpages/methamphetamine.html.

To find publicly funded treatment facilities by State, visit www.findtreatment.samhsa.gov.

For street terms searchable by drug name, street term, cost and quantities, drug trade, and drug use, visit www.whitehousedrugpolicy.gov/streetterms/default.asp.

** These data are from the 2008 Monitoring the Future survey, funded by the National Institute on Drug Abuse, National Institutes of Health, Department of Health and Human Services, and conducted by the University of Michigan's Institute for Social Research. The study has tracked 12th-graders' illicit drug abuse and related attitudes since 1975; in 1991, 8th- and 10th-graders were added to the study. The latest data are online at www.drugabuse.gov.*

*** "Lifetime" refers to use at least once during a respondent's lifetime. "Past year" refers to use at least once during the year preceding an individual's response to the survey. "Past month" refers to use at least once during the 30 days preceding an individual's response to the survey.*

**** NSDUH (formerly known as the National Household Survey on Drug Abuse) is an annual survey of Americans age 12 and older conducted by the Substance Abuse and Mental Health Services Administration. Copies of the latest survey are available at www.samhsa.gov and from NIDA at 877-643-2644.*

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Appendix F

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Methamphetamine enters brain quickly and lingers

First study of methamphetamine uptake, distribution, and clearance in humans helps explain why the drug is so addictive and damaging to brain

UPTON, NY — Using positron emission tomography (PET) to track tracer doses of methamphetamine in humans' brains, scientists at the U.S. Department of Energy's (DOE) Brookhaven National Laboratory find that the addictive and long-lasting effects of this increasingly prevalent drug can be explained in part by its pharmacokinetics — the rate at which it enters and clears the brain, and its distribution. This study in 19 healthy, non-drug-abusing volunteers includes a comparison with cocaine and also looked for differences by race. It will appear in the November 1, 2008, issue of *NeuroImage*.

"Methamphetamine is one of the most addictive and neurotoxic drugs of abuse," said Brookhaven chemist Joanna Fowler, lead author on the study. "It produces large increases in dopamine, a brain chemical associated with feelings of pleasure and reward — both by increasing dopamine's release from nerve cells and by blocking its reuptake."

Studies by Fowler and others have shown that drugs that produce greater elevations in brain dopamine tend to be more addictive. But other factors, including the speed with which a drug enters and clears the brain and its distribution within the brain, can also be important in determining its addictive and toxic potential.

In undertaking this first study of methamphetamine pharmacokinetics, the researchers also wanted to know if there were differences between Caucasians and African Americans. "Reports that the rate of methamphetamine abuse among African Americans is lower than for Caucasians led us to question whether biological or pharmacokinetic differences might explain this difference," Fowler said.

The scientists measured brain uptake, distribution, and clearance of methamphetamine by injecting 19 normal healthy men (9 Caucasian, 10 African American) with a radioactively tagged form of the drug in "trace" doses too small to have any psychoactive effects. They used PET scanning cameras to monitor the concentration and distribution of the tagged methamphetamine in the subjects' brains. On the same day, the same subjects were injected with trace doses of cocaine and scanned for comparison. The scientists also used PET to measure the number of dopamine reuptake proteins, known as dopamine transporters, available in each research subject's brain.

Like cocaine, methamphetamine entered the brain quickly, a finding consistent with both drugs' highly reinforcing effects. Methamphetamine, however, lingered in the brain significantly longer than cocaine, which cleared quickly. In fact, some brain regions, particularly white matter, still showed signs of tracer methamphetamine at the end of the 90-minute scanning session, by which time all cocaine had been cleared. The distribution of methamphetamine in the brain was remarkably different from that of cocaine. Whereas cocaine was concentrated only in the 'reward' center and cleared rapidly, methamphetamine was concentrated all over the brain, where it remained throughout the study.

"This slow clearance of methamphetamine from such widespread brain regions may help explain why the drug has such long-lasting behavioral and neurotoxic effects," Fowler said. Methamphetamine is known to produce lasting damage not only to dopamine cells but also to other brain regions, including white matter, that are not part of the dopamine network.

Surprisingly, the researchers found significant differences in cocaine pharmacokinetics between African Americans and Caucasians, with the African Americans exhibiting higher uptake of cocaine, a later rise to peak levels, and slower clearance. In contrast, the scientists found no differences in methamphetamine pharmacokinetics between these groups.

"This suggests that variables other than pharmacokinetics and bioavailability account for the lower prevalence of methamphetamine abuse in African Americans," Fowler said. "The differences observed for cocaine pharmacokinetics are surprising considering there are no differences in cocaine abuse prevalence between these two ethnic groups." These differences may merit further study, and also suggest the need to match subjects by ethnic group in future studies to avoid interference from this potentially confounding variable.

Another interesting finding was that across all research subjects, the level of dopamine transporters was directly related to the level of methamphetamine taken up by the brain. This finding suggests that transporter proteins somehow play a role in regulating the brain's uptake of this drug.

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This research was funded by the National Institute on Drug Abuse, the National Institute on Alcohol Abuse and Alcoholism Intramural Program, and by the Office of Biological and Environmental Research within DOE's Office of Science. Brain-imaging studies such as PET are a direct outgrowth of DOE's long-standing investment in basic research in chemistry, physics, and nuclear medicine. The ongoing neuroimaging research at Brookhaven is a prime example of how DOE's national laboratories bring together the expertise of chemists, physicists, and medical scientists to address questions of profound significance for society.

One of ten national laboratories overseen and primarily funded by the Office of Science of the U.S. Department of Energy (DOE), Brookhaven National Laboratory conducts research in the physical, biomedical, and environmental sciences, as well as in energy technologies and national security. Brookhaven Lab also builds and operates major scientific facilities available to university, industry and government researchers. Brookhaven is operated and managed for DOE's Office of Science by Brookhaven Science Associates, a limited-liability company founded by the Research Foundation of State University of New York on behalf of Stony Brook University, the largest academic user of Laboratory facilities, and Battelle, a nonprofit, applied science and technology organization.

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Appendix G

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Neurotoxicity of Substituted Amphetamines: Molecular and Cellular Mechanisms

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(Submitted 06 November 2006; In final form 11 January 2007)

The amphetamines, including amphetamine (AMPH), methamphetamine (METH) and 3,4-methylenedioxymethamphetamine (MDMA), are among abused drugs in the US and throughout the world. Their abuse is associated with severe neurologic and psychiatric adverse events including the development of psychotic states. These neuropsychiatric complications might, in part, be related to drug-induced neurotoxic effects, which include damage to dopaminergic and serotonergic terminals, neuronal apoptosis, as well as activated astroglial and microglial cells in the brain. The purpose of the present review is to summarize the toxic effects of AMPH, METH and MDMA. The paper also presents some of the factors that are thought to underlie this toxicity. These include oxidative stress, hyperthermia, excitotoxicity and various apoptotic pathways. Better understanding of the cellular and molecular mechanisms involved in their toxicity should help to generate modern therapeutic approaches to prevent or attenuate the long-term consequences of amphetamine use disorders in humans.

Keywords: Substituted amphetamines; Methamphetamine; Methylenedioxyamphetamine; MDMA; Serotoninergic neurons; Dopaminergic neurons; Hyperthermia; Neurotoxicity

AMPHETAMINE

Amphetamine (AMPH) is a psychostimulant that belongs to widely used illegal drugs in the world. AMPH is a popular drug of abuse in Australia (Bartu *et al.*, 2004), Belgium (Raes and Verstraete, 2005), Brazil (Silva and Yonamine, 2004), Switzerland (Augsburger *et al.*, 2005) and UK (Wylie *et al.*, 2005). AMPH is a common drug of abuse in Sweden and other northern European countries (Jones, 2005; Gustavsen *et al.*, 2006). In the USA, non-medical use of medications prescribed for ADHD treatment, including those that contain AMPH, is high among high school and college students (McCabe *et al.*, 2004; 2005). It has been reported that the abuse of these drugs is second only to marijuana (Brown *et al.*, 2001).

AMPH abuse is associated with very serious harms. These include increased psychological morbidity, dependence and health problems. For example, acute AMPH side-effects include tachycardia, hypertension, hyperthermia, increased muscle tention, liver and renal failure, nausea, blurred vision, ataxia, anxiety, psychosis and seizures (Kalant and Kalant, 1975; Janowsky and Risch, 1979; Alldredge *et al.*, 1989; Murray, 1998). Other severe and fatal AMPH intoxications have also been reported (Ginsberg *et al.*, 1970; Kalant and Kalant, 1975; Salanova and Taubner, 1984; De

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Letter *et al.*, 2006; Steentoft *et al.*, 2006). Chronic AMPH abuse is associated with impairments in attention and memory, problems with learning, as well as compromised decision making (McKetin and Mattick, 1997; Rogers *et al.*, 1999; Ornstein *et al.*, 2000). Some of these neuropsychiatric complications are thought to be related to AMPH-induced neurotoxic effects which consist of decreases in tyrosine hydroxylase (TH) activity (Ellison *et al.*, 1978), long-term dopamine (DA) depletion (Wagner *et al.*, 1980a), loss of dopamine transporters (DAT) (Scheffel *et al.*, 1996; Krasnova *et al.*, 2001), as well as decreases in vesicular monoamine transporter proteins (Krasnova *et al.*, 2001). In addition to its effects on monoaminergic terminals, AMPH can cause cell death of primary cortical cells, TH-positive mesencephalic neurons, and of PC12 cells *in vitro* (Stumm *et al.*, 1999; Lotharius and O'Malley, 2001; Oliveira *et al.*, 2002) as well as degeneration of cell bodies in the cortex of AMPH-treated rodents (Jakab and Bowyer, 2002). The drug can also cause the activation of caspase-3 and appearance of TUNEL-positive cells in the striatum (Krasnova *et al.*, 2005). Calbindin- and DA- and cAMP-regulated phosphoprotein, Mr 32 kD (DARPP-32)-positive medium spiny projection neurons, but not choline acetyltransferase (ChAT)-, parvalbumin- or somatostatin-positive interneurons undergo AMPH-induced apoptosis (Krasnova *et al.*, 2005). Although the mechanisms for AMPH-mediated toxicity are not completely clear, they appear to include uptake into DA terminals, DA release, oxidative stress and the activation of p53-dependent and mitochondria-mediated cell death pathways. Herein, the data supporting these mechanisms in AMPH toxicity are reviewed.

AMPH Toxicity Involves ROS Formation and ROS-mediated Transcriptional Changes. AMPH-induced redistribution of DA from synaptic vesicles to the cytosol followed by its release to the extracellular space by reverse transport through DAT causes increased DA levels in the synaptic cleft (Sulzer *et al.*, 1995). DA metabolism is accompanied by the production of hydroxyl (Huang *et al.*, 1997) and superoxide (Krasnova *et al.*, 2001) radicals that participate in the toxic effects of the drug via free radical-mediated destruction of monoaminergic terminals (Huang *et al.*, 1997; Cadet and Brannock, 1998; Wan *et al.*, 2000; Krasnova *et al.*, 2001). This occurs because reactive oxygen species (ROS) induced by AMPH administration can exceed the compensating abilities of antioxidant enzymes such as superoxide dismutases (SODs), catalase and glutathione peroxidase (Cadet and Brannock, 1998). The possible involvement of superoxide radicals in AMPH toxicity is also supported by the findings that transgenic mice that overexpress CuZnSOD are partially protected against the toxic effects of the drug on dopaminergic systems (Krasnova *et al.*, 2001).

Because ROS play a role in cellular signaling processes, including the regulation of transcriptional factors (Poli *et al.*, 2004), induction or suppression of transcription factors with subsequent activation or repression of genes that encode proteins involved in various neuronal functions might be critical steps in AMPH-induced cascades of toxic events. These ideas are supported by the demonstration that administration of AMPH causes activation of AP-1 transcription factors (Persico *et al.*, 1995; Ferguson *et al.*, 2003; Milanovic *et al.*, 2006). The possibility that superoxide radicals might be involved in AMPH-induced transcriptional responses has been tested using microarray analyses (Krasnova *et al.*, 2002). This allowed the identification of 37 genes that show superoxide-mediated responses. Among these are genes that belong to classes of transcription factors, growth factors, heat shock proteins (HSPs), and xenobiotic metabolism. In response to neuronal damage, organisms initiate and elaborate events that trigger neuroprotective pathways that serve to minimize or prevent damage; they also function to increase the chance of functional recovery (Wieloch and Nikolich, 2006). These pathways include the increased synthesis and release of growth factors and cytokines such as the neuronal protein, activin A (Werner and Alzheimer, 2006), which is activated by AMPH in a superoxide-responsive manner (Krasnova *et al.*, 2002). The participation of activin A in protective mechanisms is illustrated by the reports that it reduces MPP^+-induced cellular damage to DA neurons *in vitro* (Krieglstein *et al.*, 1995) and rescues striatal neurons from excitotoxic lesioning with quinolic acid (Hughes *et al.*, 1999). Another AMPH-responsive superoxide-mediated gene is macrophage colony-stimulating factor which is involved in the pro-

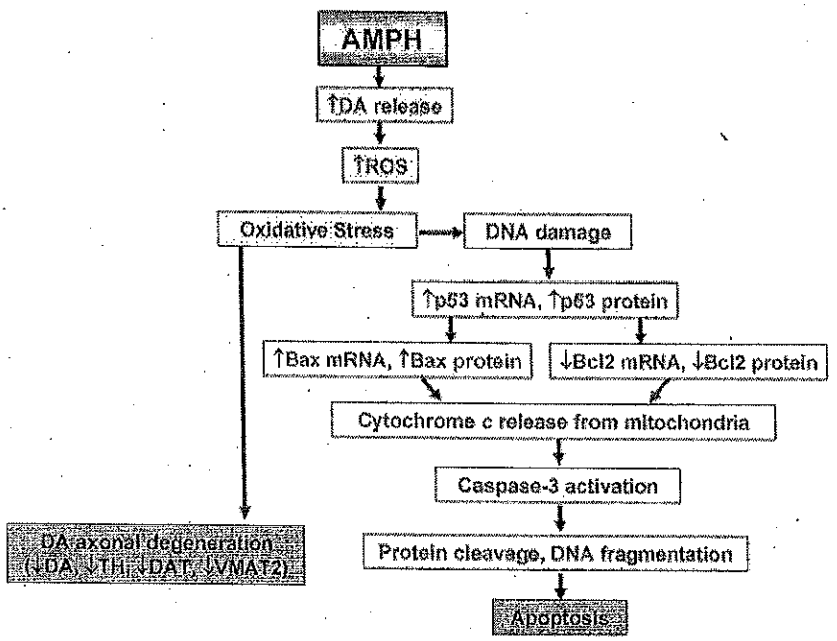

FIGURE 1 Overview of the molecular mechanisms involved in AMPH neurotoxicity. Oxidative stress, p53 and mitochondrial pathway play an essential role in the AMPH-induced neuronal apotosis and DA terminal degeneration.

liferation and migration of activated microglia into injured sites of the brain (Imai and Kohsaka, 2002). Additional genes whose transcript levels are induced by AMPH code for HSPs such as HSP110 and HSC70. HSPs have been shown to protect cells against oxidative damage (Papp *et al.*, 2003; Macario and Conway de Macario, 2005).

AMPH Toxicity Involves Activation of the p53-mediated Cell Death Pathway.

ROS-induced stresses are known to be associated with DNA damage and p53 accumulation *in vitro* (Lombard *et al.*, 2005). P53 activation has been shown to participate in events that cause neuronal apoptosis (Culmsee and Mattson, 2005). This is thought to be related to the influence exerted by p53 on the expression of the Bcl-2 family of proteins which include the pro-apoptotic protein, Bax and the anti-apoptotic protein, Bcl-2 (Moll *et al.*, 2005; Chowdhury *et al.*, 2006). Specifically, p53 causes upregulation of Bax and downregulation of Bcl-2 (Moll *et al.*, 2005; Chowdhury *et al.*, 2006). As reported above, AMPH has been shown to cause neuronal cell death in various brain regions (Jakab and Bowyer, 2002; Krasnova *et al.*, 2005). The AMPH-induced neuronal apoptosis has been recently shown to involve the activation of the p53 pathway with secondary increases in Bax levels and decreases in Bcl-2 levels in the mouse striatum (Krasnova *et al.*, 2005). The role of Bax activation in AMPH-related apoptosis was further supported by experiments showing that Bax-deficient mice were partially resistant to drug-induced cell death (Krasnova *et al.*, 2005). Figure 1 shows a schematic representation of the mechanisms that may underlie AMPH-related apoptosis and DA terminal degeneration.

AMPH Treatment and Temperature Regulation

Temperature regulation appears to be also an important factor in the toxic responses to AMPH. In rodents, the psychostimulant was shown to produce biphasic effects with low doses (≤ 2.5 mg/kg) inducing hypothermia and higher doses (≥ 5 mg/kg) causing hyperthermia at ambient temperature above 20°C (Seale *et al.*, 1985; Krasnova *et al.*, 2001; Baker and Meert, 2003). This effect was found to be dose-dependent, with the degree of hyperthermia correlating to AMPH and DA levels in rat striatal microdialysate (Clausing and Bowyer, 1999). Several studies have also hinted to connections between hyperthermic and neurotoxic

actions of AMPH (Clausing *et al.*, 1995; Miller and O'Callaghan, 1996). Conditions that reduce or prevent AMPH-induced increases in core body temperature are, at least, partially neuroprotective (Clausing *et al.*, 1995; Miller and O'Callaghan, 1996). In contrast, hyperthermia could exacerbate AMPH toxicity because the formation of free radicals in the brain is elevated by temperature increase (Kil *et al.*, 1996) and because hyperthermia also potentiates the cytotoxic effects of ROS (Lin *et al.*, 1991). These ideas are also supported by the report that hyperthermia significantly increases DA quinone formation (LaVoie and Hastings, 1999) since quinones derived from DA have the ability to inhibit proteasome (Zafar *et al.*, 2006) which is involved in detoxification mechanisms.

Although AMPH-induced increases in temperature are thought to be involved in the toxicity of the drug, the manner by which the temperature is induced remains to be determined. DA release (Clausing and Bowyer, 1999) and D_1 receptor stimulation (Sanchez, 1989; Zarrindast and Tabatabai, 1992; Verma and Kulkarni, 1993) have been implicated. The observations that animals with severe hyperthermia released more DA in the striatal extracellular space provide partial support for this contention (Clausing and Bowyer, 1999). Moreover, the idea is also supported by the reports that hyperthermia is induced by administration of the D_1 agonist SKF 38393 in mice (Sanchez, 1989; Zarrindast and Tabatabai, 1992; Verma and Kulkarni, 1993), the effect that could be blocked by D_1 antagonist SCH 23390 (Sanchez, 1989; Zarrindast and Tabatabai, 1992). It has to be pointed out that since AMPH can cause release of other monoamines (Seiden *et al.*, 1993), their possible involvement also needs to be considered. For example, lesions of ventral norepinephrine bundle innervating the hypothalamus and limbic system cause attenuation of AMPH-induced hyperthermia in rats (Kostowski *et al.*, 1982).

In addition to DA release, AMPH-induced production of free radicals might also contribute to thermal instability (Krasnova *et al.*, 2001). Mice that overexpress the antioxidant enzyme, CuZnSOD, in the brain show no hyperthermic responses after AMPH treatment and are protected against long-term neurotoxic drug effects (Krasnova *et al.*, 2001). Thus, the possibility of complex interactions between thermoregulation and free radical load in the long-term neurotoxicity induced by this illicit drug needs to be considered.

It is also of interest to note that various strains of mice show different hyperthermic responses to AMPH. Specifically, psychostimulants caused substantial hyperthermia in CD-1 (Krasnova *et al.*, 2001), Swiss-Webster (Craig and Kupferberg, 1972), DBA/2 (Seale *et al.*, 1985), and BALB/c mice (Jori and Rutczynski, 1978), while C57BL/6 (Seale *et al.*, 1985; Krasnova *et al.*, 2001) and C_3H animals (Jori and Rutczynski, 1978) had low to moderate transient temperature increase. These differences in temperature responses may help to further dissect the role of hyperthermia in AMPH toxicity. For example, it seems there is no simple algorithm to predict toxicity based on temperature responses because CD-1 mice are more resistant to AMPH neurotoxicity than C57BL/6 mice in spite of showing greater and longer-lasting hyperthermia than C57BL/6 mice (Krasnova *et al.*, 2001).

Finally, the issues of temperature regulation have major clinical implications because AMPH can cause fatal hyperpyrexia in humans (Ginsberg *et al.*, 1970; Kalant and Kalant, 1975; Callaway and Clark, 1994; De Letter *et al.*, 2006). Thus, understanding of the root causes of AMPH-induced hyperthermia might help to develop therapeutic approaches that can prevent or attenuate the disastrous effects of this drug when taken in high doses.

METHAMPHETAMINE

Metamphetamine (METH, Speed, crank) is abused worldwide due to its powerful stimulant properties that cause the user to feel "high" and to have increased energy (McCann *et al.*, 1998b; Sekine *et al.*, 2001; Farrell *et al.*, 2002). METH is easily available because it can be synthesized cheaply and distributed to various communities throughout the world. Presently, there is widespread abuse in the United States where it has migrated from the West Coast to other states (Puder *et al.*, 1988; Derlet *et al.*, 1989; Cho and Melega, 2002). METH can be abused via multiple routes which include oral, intravenous and smoking administration. In addition to its euphorigenic effects, METH can also cause anxiety, increased agitation, delirium, psychotic states, cognitive and psychomotor impairments, seizures, and death (Wilson *et al.*, 1996;

Lan *et al.*, 1998; Buffenstein *et al.*, 1999; Yui *et al.*, 1999; Simon *et al.*, 2000; Volkow *et al.*, 2001a; London *et al.*, 2004; Dore and Sweeting, 2006). Cerebral vasculitis, cerebrovascular accidents due to hemorrhage or vasospasm, and cerebral edema have also been reported in METH abusers (Chynn, 1968; Salanova and Taubner, 1984). The drug can also cause neurodegenerative changes in the brains of human addicts. These pathological changes include loss of striatal DAT observed in positron emission tomographic (PET) studies (Volkow *et al.*, 2001b; Sekine *et al.*, 2003) and in post-mortem investigations, loss of serotonin transporters (5-HTT) (Sekine *et al.*, 2006), decrease in the levels of DA, serotonin (5-HT) and their metabolites (Wilson *et al.*, 1996). A number of studies have documented that METH can cause long-term damage to presynaptic dopaminergic and serotonergic terminals in rodents (Ricaurte *et al.*, 1980; Wagner *et al.*, 1980b). More recently, it has also been shown that the drug can cause death of cell bodies both *in vitro* (Cadet *et al.*, 1997; Stumm *et al.*, 1999) and *in vivo* (Eisch and Marshall, 1998; Deng *et al.*, 1999; Deng and Cadet, 2000; O'Dell and Marshall, 2000). In what follows, we discuss some of the mechanisms that have been implicated in METH-induced neurodegenerative effects.

Role of Oxidative Stress in METH-induced Toxicity

The biochemical actions of the drug depend on its entry into monoaminergic terminals (Berger *et al.*, 1992; Iversen, 2006), followed by entry into monoaminergic vesicle consequent to its interaction with vesicular monoamine transporters (Sulzer *et al.*, 1995). This is followed by displacement of monoamines into the cytoplasm of the terminals and METH-induced monoamine release into respective synaptic clefts (Baldwin *et al.*, 1993; Marshall *et al.*, 1993; Cubells *et al.*, 1994; Sulzer *et al.*, 1995; Schwartz *et al.*, 2006). METH neurotoxicity appears to depend on both, DA released within terminals and on DA released in synaptic clefts (Cadet and Brannock, 1998). These suggestions are supported by reports that DAT knockout mice are resistant to METH-induced degeneration of DA axons (Fumagalli *et al.*, 1998) and by observations that psychostimulant toxicity depends on quinone formation consequent to increased DA levels within nerve terminals (LaVoie and Hastings, 1999). METH-related quinone formation is thought to be associated with the generation of superoxide radicals and hydrogen peroxide during quinone redox cycling (Stokes *et al.*, 1999; Miyazaki *et al.*, 2006). A role for oxidative mechanisms in the neurotoxic effects of the drug is supported by observations that administration of *N*-acetyl-L-cysteine, ascorbic acid or vitamin E was able to protect against METH-induced destruction of monoaminergic terminals (Wagner *et al.*, 1985; De Vito and Wagner, 1989; Fukami *et al.*, 2004). In addition, selenium and melatonin can also provide protection against METH toxicity (Ali *et al.*, 1999; Imam and Ali, 2000). The participation of superoxide radicals in the neurotoxic effects of METH on DA nerve terminals was tested by injecting METH to transgenic mice that overexpress the human CuZnSOD gene (Cadet *et al.*, 1994a; Hirata *et al.*, 1996; Jayanthi *et al.*, 1998). These mice have much higher CuZnSOD activity than wild-type animals from similar backgrounds (Jayanthi *et al.*, 1998; Jayanthi *et al.*, 1999) and were indeed protected against the toxic effects of the drug. In contrast, inhibition of SOD by diethyldithiocarbamate potentiates the nefarious effects of METH (De Vito and Wagner, 1989). Furthermore, bromocriptine, which scavenges hydroxyl radicals, was also able to attenuate METH-induced DA depletion in mice (Kondo *et al.*, 1994). When taken together, these observations support the notion that DA release caused by METH is accompanied by redox cycling of dopaquinone and consequent formation of oxygen-based radicals such as superoxide radicals. Reports that METH can induce changes in the levels of glutathione (Harold *et al.*, 2000) and of antioxidant enzymes (Jayanthi *et al.*, 1998), increase lipid peroxidation (Jayanthi *et al.*, 1998; Gluck *et al.*, 2001), and induce the formation of protein carbonyls (Gluck *et al.*, 2001) provide further support for the thesis that oxygen-based radicals are involved in METH-induced toxicity (Cadet and Brannock, 1998).

METH Toxicity and Excitotoxicity

METH-induced neurotoxicity also appears to occur via excitotoxic damage secondary to glutamate

release and activation of glutamate receptors. Glutamate toxicity is dependent, in part, on the production of nitric oxide (NO) (Dawson and Dawson, 1998; Chung *et al.*, 2005). The idea of the involvement of glutamate in METH toxicity is supported by observations that METH can cause glutamate release in the brain (Nash *et al.*, 1988; Baldwin *et al.*, 1993; Marshall *et al.*, 1993; Abekawa *et al.*, 1994; Mark *et al.*, 2004). In addition, some glutamate antagonists have been shown to attenuate METH-induced dopaminergic toxicity (Sonsalla *et al.*, 1989; Battaglia *et al.*, 2002) (see later discussion on temperature). Glutamate-mediated NO formation appears to also be involved in METH toxicity because knockout mice that are deficient in either neuronal (nNOS) or inducible (iNOS) nitric oxide synthase (NOS) are resistant to drug-induced toxic damage to monoaminergic terminals (Itzhak *et al.*, 1998). These data have solidified the argument for a role of the glutamate/NO pathway in METH neurotoxicity (Itzhak *et al.*, 1998; Imam *et al.*, 2001; Itzhak and Ali, 2006). Finally, various nNOS inhibitors, which do not affect hyperthermia, can also protect against destruction of monoaminergic axons caused by METH administration (Itzhak *et al.*, 2000; Sanchez *et al.*, 2003). In addition to their roles in the damage of monoaminergic terminals, oxygen-based radicals and NO appear to be involved in METH-related cell death because CuZnSOD transgenic mice show partial protection against drug-induced apoptosis (Deng and Cadet, 2000). Moreover, death of rat fetal mesencephalic cells caused by METH treatment was abrogated by the use of NOS inhibitors (Sheng *et al.*, 1996).

Role of Thermal Instatibility in METH Toxicity

There is substantial evidence that hyperthermia participates in METH-induced toxicity on monoaminergic systems. Manipulations that result in higher temperatures cause increases in METH toxicity, whereas those that decrease temperatures have been shown to provide some degree of protection (Bowyer *et al.*, 1994; Miller and O'Callaghan, 1994; Albers and Sonsalla, 1995; Farfel and Seiden, 1995). The potentiative effects of hyperthermia might occur through increased formation of DA-dependent reactive oxygen species. In contrast, there are pharmacological agents that block METH toxicity without influencing the thermal responses in animals. For example, inhibition of nNOS blocks METH toxicity without altering the hyperthermic response (Itzhak *et al.*, 2000; Sanchez *et al.*, 2003). DA uptake blockers also protect in a fashion that appear to be independent of any effects on temperature (Callahan *et al.*, 2001).

In addition to its effects on monoaminergic terminals, METH can also cause cell death. Potential protective effects of various genetic and pharmacological manipulations have been tested in that model. For example, knockout mice that are partially deficient of c-Jun show protection against METH-induced neuronal apoptosis, an effect that is independent of hyperthermia (Deng *et al.*, 2002b). Intracerebral injection of neuropeptide Y (NPY) has recently been shown to cause attenuation of the apoptotic effects of the drug in mice (Thiriet *et al.*, 2005). Because NPY is involved in thermoregulation (Richard, 1995; Levine *et al.*, 2004) and because METH-related increases in body temperature are thought to participate in METH toxicity (Cadet *et al.*, 2003, for review), the possibility that NPY might have prevented drug-induced hyperthermia was tested (Thiriet *et al.*, 2005). NPY was found to attenuate body temperature increases after the second of the four METH injections but not during the later phases of hyperthermia (Thiriet *et al.*, 2005). These observations suggest that NPY-induced protection is, in part, dependent on its effects on body temperature. It appears that METH-related changes in body temperature participate, but are not essential in the manifestations of drug toxicity.

Microglial Reactions and METH Toxicity

Microglial cells are the major immunocompetent cells in the brain. They express chemokines, cytokines and their receptors. Under normal conditions, these cells provide extensive and continuous surveillance of their cellular environment (Raivich, 2005). Microglial cells are activated by various types of pathological states including infectious processes (Rock *et al.*, 2004) and neural injuries (Ladeby *et al.*, 2005). This activation includes dramatic changes in appearance, migration to the site of the damage, and phagocytosis of dying and dead cells. Microglia can also produce small signaling molecules, called cytokines, to trigger astrocytes to respond to the injury site. Recently,

reactive microgliosis has been implicated in a number of neurological disorders including Alzheimer's (Xiang *et al.*, 2006) and Huntington's (Sapp *et al.*, 2001) diseases.

Evidence accumulating from several laboratories has recently implicated reactive microglial cells as culprits in the manifestation of METH toxicity. Asanuma *et al.* (2003) reported that the non-steroidal anti-inflammatory drug, ketoprofen, caused protection against METH-induced dopaminergic toxicity and suppressed drug-mediated microgliosis. Thomas and colleagues (2004) subsequently reported that METH caused dose-dependent microglial activation which coincided with DA terminal degeneration. LaVoie *et al.* (2004) have also provided evidence that microgliosis precedes METH-induced pathological states in striatal dopaminergic terminals. More importantly, manipulations such as the use of MK-801 and dextromethorphan which protect against METH toxicity also inhibit microglial activation (Thomas and Kuhn, 2005). In contrast, minocycline has been reported to block microglial activation without providing protection against METH-induced damage (Sriram *et al.*, 2006). Microglial cells might potentiate drug-related damage by releasing toxic substances such superoxide radicals and NO which have already been implicated in METH neurotoxicity (see discussion above). When taken together, these observations suggest that identifying the specific role that microglial cells play in DA terminal degeneration might help to develop specific therapeutic approaches for patients who have been exposed to METH.

Involvement of AP-1 Related Transcription Factors in METH-induced Neurotoxicity

The accumulated evidence had suggested that some effects of oxygen-based radicals might be mediated by activation of AP-1 transcription factors (Dalton *et al.*, 1999). Tests for the possibility that METH toxicity might also be associated with variations in the expression of these proteins have revealed changes in the expression of a number of AP-1 related genes within 2 hours after drug administration (Cadet *et al.*, 2001). These include up-regulation of c-jun, c-fos, jun B, as well as jun D (Cadet *et al.*, 2001). These changes are probably related to METH-induced generation of free radicals. ROS such as hydroxyl and superoxide radicals can induce the expression of many genes via their regulation of AP-1 transcription factors (Dalton *et al.*, 1999). The role for c-fos in METH-induced neuropathological changes has been confirmed by using c-fos +/- mice which show increased degeneration of DA terminals and increased cell death after psychostimulant treatment (Deng *et al.*, 1999). These observations suggest a protective role for c-fos against METH damage. Some of the factors that might be involved in causing this partial protection include integrins that belong to cell adhesion receptors and are also involved in the regulation of signal transduction (Gilcrease, 2006). This idea is supported by the evidence of decreased basal levels of integrin expression in c-fos +/- mice and the further reduction of these receptors in response to toxic doses of METH (Betts *et al.*, 2002). This conclusion is further supported by the observations that integrins can promote cell survival after injury and apoptotic insults via signaling through the PI3K-Akt pathway which leads to BAD phosphorylation, therefore reducing BAD ability to block the anti-apoptotic effects of Bcl-2 (Martin and Vuori, 2004; Gilcrease, 2006). In contrast, inhibition of integrins increases apoptotic cell death (Martin and Vuori, 2004; Gilcrease, 2006).

Because c-jun knockout mice show partial protection against the adverse effects of METH (Deng *et al.*, 2002b), it is likely that c-jun is involved in the pro-death effects of the drug. Moreover, because the c-jun knockout mice and their wild-type counterparts show similar degree of METH-induced dopaminergic toxicity, c-jun appears to only be involved in the mediation of neuronal apoptosis in cells postsynaptic to DA terminals.

Role of DNA Damage in METH-induced Toxicity

As mentioned above, METH has been shown to cause neuronal apoptosis in several brain regions (Deng *et al.*, 2001). Because apoptosis is associated with DNA damage, it was thought possible that administration of the drug might trigger responses meant to repair the METH-induced DNA damage. Microarray analyses have indeed revealed that METH administration caused changes in the expression of several genes that participate in DNA repair processes (Cadet *et al.*, 2002). These changes

are probably related to METH-induced prooxidant states because oxidative stress can cause single and double DNA strand breaks (Li and Trush, 1993). These breaks can be repaired via base excision repair (BER), nucleotide excision repairs (NER), mismatch repair (MMR), and DNA damage reversal (Petit and Sancar, 1999; Hsieh, 2001; Nilsen and Krokan, 2001). Thus, the observations that METH treatment can cause upregulation of APEX, PolB, and LIG1 suggest that these changes might be compensatory increases aimed at counteracting METH-mediated ROS-induced DNA damage through the BER pathway. If the psychostimulant can cause similar DNA damage in humans, these observations might offer a partial explanation for the developmental abnormalities observed in babies born of METH abusing mothers (Smith *et al.*, 2006).

Involvement of Mitochondrial Death Pathway in METH-induced Apoptosis

Another interesting group of proteins that are differentially regulated by METH includes Bcl-2 family (Stumm *et al.*, 1999; Cadet *et al.*, 2001; Jayanthi *et al.*, 2001). Specifically, METH caused upregulation of pro-apoptotic proteins, BAX and BID, and downregulation of the anti-death proteins, Bcl-2 and Bcl-X_L. The changes in pro-death proteins are consistent with observations that METH administration is associated with release of mitochondrial contents into the cytosol (Deng *et al.*, 2002a; Jayanthi *et al.*, 2004). These include cytochrome *c* and apoptosis inducing factor (AIF). When taken together with the recent *in vitro* demonstration that METH can cause release of cytochrome *c* from mitochondria, activation of caspases 9 and 3, as well as activation of DFF40 and its transit to the nucleus (Deng *et al.*, 2002a), the *in vivo* data implicate a formal role of mitochondria in METH-induced neuronal degeneration. Other factors released from mitochondria such as Smac/DIABLO, endonuclease G, and AIF also participate in dismantling cells during apoptosis (Ravagnan *et al.*, 2002). These proteins, including AIF and Smac/DIABLO, have now been shown to be involved in METH-induced apoptosis (Jayanthi *et al.*, 2004). Their release is followed by activation of caspase 3 and the breakdown of several structural cellular proteins (Jayanthi *et al.*, 2004). Thus, these observations implicate the mitochondrial death pathway as a major player in METH-related cell death in the rodent brain (Cadet *et al.*, 2005). This suggestion is supported by the fact that overexpression of Bcl-2 can protect against drug-induced apoptosis (Cadet *et al.*, 1997).

Involvement of the Endoplasmic Reticulum (ER)-dependent Death Pathway in METH-induced Apoptosis

In addition to its effects on mitochondria, METH-induced oxidative stress appears to also cause dysfunctions of other organelles such as the endoplasmic reticulum (ER) (McCullough *et al.*, 2001). The ER helps to maintain cellular homeostasis by regulating calcium signaling (Ferri and Kroemer, 2001). Dysregulation of intracellular calcium homeostasis can cause ER stress and ER-mediated apoptosis (Paschen, 2001). ER stress and calcium dysregulation appear to participate in METH-induced cell death because apoptotic doses of the drug can cause activation of calpain, a calcium-responsive cytosolic cysteine protease (Murachi *et al.*, 1980), which is involved in ER-dependent cell death (Nakagawa and Yuan, 2000). A role for the ER in METH toxicity is supported by the fact that apoptotic doses of METH (Jayanthi *et al.*, 2004) also influence the expression of proteins, such as caspase-12, GRP78/BiP (glucose-regulated protein/immuno-globulin heavy chain binding protein) and CHOP/GADD153 (C/EBP homology protein/growth arrest and DNA damage 153) that participate in ER-induced apoptosis (Zinszner *et al.*, 1998). The observed ER stress in METH-induced neurotoxicity might be secondary, in part, to direct effects of the psychostimulant (Asanuma *et al.*, 2000), to METH-mediated oxidative stress (Cadet *et al.*, 1994a; Cadet and Brannock, 1998; Jayanthi *et al.*, 1998), and to shifts in BAX/Bcl-2 ratios induced by the drug (Jayanthi *et al.*, 2001).

Involvement of the Fas/Fas Ligand Death Pathway in METH-induced Apoptosis

In addition to the mitochondrial death pathway, cell death can occur consequent to activation of Fas receptors by Fas ligand (FasL) (Barnhart *et al.*, 2003; Choi and Benveniste, 2004). FasL (TNFSF6) (Li-Weber *et al.*, 1999; Li-Weber and Krammer, 2002; Droin *et al.*, 2003) is a member of the TNF superfamily of cytokines (Locksley *et al.*, 2001) and is involved in causing apoptosis in various models

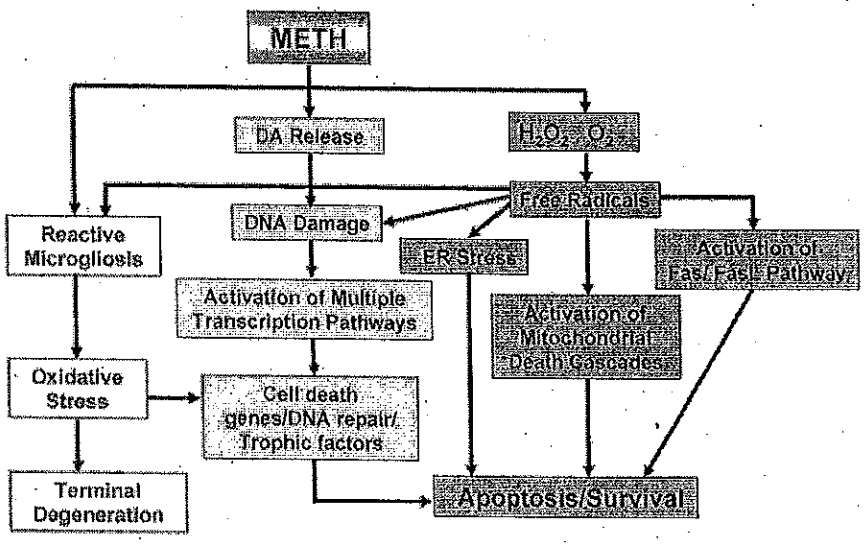

FIGURE 2 METH-regulated molecular events that lead to neuronal apoptosis and terminal degeneration in the striatum. This figure summarizes findings of the various papers that have addressed the issue of METH-induced neurotoxicity in the mammalian brain. The data indicate that oxidative mechanisms and cell death pathways are involved in the manifestation of METH toxicity.

of neuronal injury (Qiu *et al.*, 2002). METH was shown to increase the expression of FasL (Jayanthi *et al.*, 2005; reviewed in Cadet *et al.*, 2005). It was also shown that METH can induce cleavage of caspase 8, which is a known participatant in the Fas death pathway (Nagata, 1999).

We have summarized these molecular mechanisms in a theoretical scheme that represents the sequence of events leading to METH-induced neuronal apoptosis and terminal degeneration (Fig. 2).

METHYLENEDIOXYMETHAMPHETAMINE (MDMA, Ecstasy)

3,4-Methylenedioxymethamphetamine (MDMA, Ecstasy) is an abused ring-substituted phenyl-isopropylamine that is related to both amphetamines and hallucinogens (McKenna and Peroutka, 1990). MDMA effects which include increased locomotor activity (Matthews *et al.*, 1989) are thought to be mediated, in part, by the release of 5-HT (Liechti *et al.*, 2000) and subsequent stimulation of its receptors (Bankson and Cunningham, 2001). In addition to MDMA behavioral effects, the drug is known to cause marked decreases in markers of 5-HT terminals (White *et al.*, 1996). Specifically, levels of 5-HT and its metabolite, 5-hydroxyindoleacetic acid (5-HIAA) (Colado and Green, 1994), tryptophan hydroxylase (TPH) activity (Stone *et al.*, 1987) and the number of 5-HT uptake sites (see Lyles and Cadet, 2003) are all decreased after MDMA administration. MDMA can also cause cell death in some *in vitro* models (Simantov and Tauber, 1997; Stumm *et al.*, 1999).

MDMA Neurotoxicity in Animals and Humans

Neurochemical and anatomical studies have shown that MDMA can cause long-term abnormalities in 5-HT systems of rodents (Schmidt *et al.*, 1986; Stone *et al.*, 1986; Commins *et al.*, 1987; Schmidt, 1987; O'Hearn *et al.*, 1988; Molliver *et al.*, 1990). These include decreased levels of 5-HT and its major metabolite, 5-HIAA (Commins *et al.*, 1987; Schmidt *et al.*, 1987; Schmidt, 1989; Molliver *et al.*, 1990), decreased number of 5-HTT (Battaglia *et al.*, 1987; Commins *et al.*, 1987; De Souza *et al.*, 1990), and decreased activity of the rate-limiting enzyme of 5-HT synthesis, TPH (De Souza *et al.*, 1990; Molliver *et al.*, 1990). These changes occur in the rodent neocortex, striatum, and hippocampus (Battaglia *et al.*, 1987; Slikker *et al.*, 1988; De Souza *et al.*, 1990; Molliver *et al.*, 1990). These abnormalities are reported to last for months or even years after drug administration (Battaglia

et al., 1988; Scanzello *et al.*, 1993; Fischer *et al.*, 1995; Lew *et al.*, 1996; Sabol *et al.*, 1996; Hatzidimitriou *et al.*, 1999).

Similar adverse effects have been reported in nonhuman primates (Ricaurte *et al.*, 1988a,b; Slikker *et al.*, 1988; Insel *et al.*, 1989; Scheffel *et al.*, 1998; McCann *et al.*, 2000). There are dose-dependent reductions in 5-HT concentrations in the cortex, caudate nucleus, putamen, hippocampus, hypothalamus and the thalamus (Ricaurte *et al.*, 1988b). Reduced 5-HT levels were evident for up to seven years following exposure to the drug (Scheffel *et al.*, 1998; Hatzidimitriou *et al.*, 1999). The MDMA-induced deficits in nonhuman primates are also reflected in the levels of 5-HIAA in the cerebrospinal fluid (CSF) (Ricaurte *et al.*, 1988a; Insel *et al.*, 1989). Living baboons treated with MDMA (5 mg/kg s.c., 2 X daily, 4 days) also show marked and prolonged decreases in 5-HTT density measured by PET imaging of (+)[^{11}C]McN-5652, a radioligand that selectively binds to the 5-HTT (Scheffel *et al.*, 1998). Brain tissues from these animals (sacrificed 3 weeks after the last PET and 13 months after MDMA administration) showed marked loss of 5-HT terminals (Scheffel *et al.*, 1998).

A number of investigators have also tested the possibility that MDMA can cause degenerative effects in the human brain (Ricaurte *et al.*, 1988a; 1990; Price *et al.*, 1989; McCann *et al.*, 1994; 1998a; 1999; Bolla *et al.*, 1998; Semple *et al.*, 1999; Gerra *et al.*, 2000; Kish *et al.*, 2000; Buchert *et al.*, 2001). Some of these studies have concluded that MDMA is also toxic to humans because CSF 5-HIAA levels are reduced in MDMA abusers (Ricaurte *et al.*, 1988a; 1990; McCann *et al.*, 1994; 1999; Bolla *et al.*, 1998). PET imaging studies, using [^{11}C]McN-5652 to selectively label 5-HTT, have reported significant differences in 5-HTT binding in MDMA abusers compared to non-MDMA users (McCann *et al.*, 1998a). 5-HTT sites were decreased in a manner that correlated with the extent of abuse (McCann *et al.*, 1998a; Ricaurte *et al.*, 2000). In a similar study, using [^{123}I]β-CIT, Reneman *et al.* (2001) investigated the effects of ecstasy abuse on the density of cortical 5-HTT. They also found decreases in cortical 5-HTT in recent MDMA abusers. However, there were no significant reductions in ecstasy abusers who had not used the drug in the past year or longer (Reneman *et al.*, 2001).

The biochemical and molecular bases of MDMA-induced neurotoxicity are being actively investigated. These pathways are thought to involve the formation of toxic MDMA metabolites, temperature dysregulation, dopamine-based quinone formation, and excitotoxic events.

Formation of Toxic Metabolites

MDMA metabolites, which generate free radicals, associated oxidative stress, and membrane damage, are thought to be involved in drug-induced neurodegeneration (Paris and Cunningham, 1992; Colado and Green, 1995). This idea is supported by observations that subcutaneous administration of MDMA metabolites, MeDA and HMA can cause decreases in 5-HT concentrations in the frontal cortex (Yeh and Hsu, 1991), although this line of research has remained controversial. The formation of hydroquinones, quinones and the subsequent generation of superoxides and hydrogen peroxide might be important to the manifestation of MDMA toxicity. These ideas are supported by the observations that the spin trap reagent and free radical scavenger, α-phenyl-*N*-*tert*-butyl nitrone (PBN), prevented MDMA-induced toxicity (Colado and Green, 1995). In addition to MDMA metabolites, the participation of a toxic metabolite of 5-HT has also been invoked because the drug causes marked increases in 5-HT release (Gudelsky and Nash, 1996; O'Shea *et al.*, 2005; Amato *et al.*, 2006).

DA-induced quinone formation is also one possible cause of MDMA toxicity. This suggestion is supported by the fact that MDMA elicits DA release (Shankaran and Gudelsky, 1998; Amato *et al.*, 2006). In addition, destruction of DA terminals by injections of 6-hydroxydopamine protects against MDMA toxicity (Schmidt *et al.*, 1990). In contrast, pretreatment with L-DOPA, which increases DA levels, exacerbates MDMA toxicity (Schmidt *et al.*, 1990). Thus, DA, which is released by MDMA into synaptic clefts, might be taken up by 5-HT terminals where it is converted into quinone by-products that damage 5-HT terminals (Schmidt and Kehne, 1990; Sprague and Nichols, 1995). It is important to point out that the DA hypothesis does not account for the fact that MDMA can damage 5-HT terminals in areas of the brain such as the hippocampus (Shankaran and Gudelsky, 1998) that are almost devoid of DA terminals and for the fact

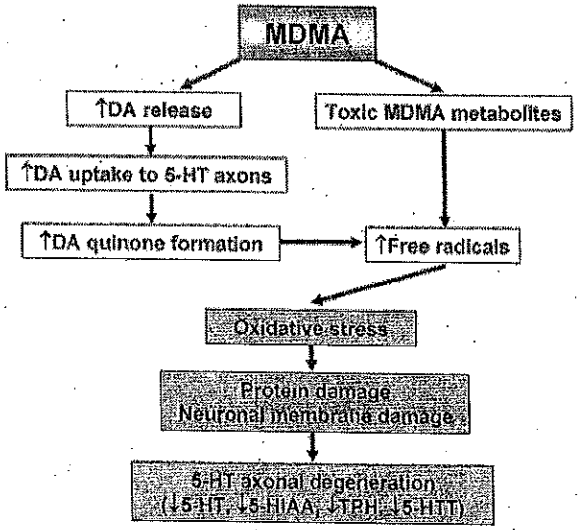

FIGURE 3 Mechanisms implicated in MDMA-induced 5-HT terminal degeneration. The schematic diagram shows that formation of toxic MDMA metabolites, DA quinones and oxidative stress may underlie MDMA toxicity towards 5-HT terminals in the brain.

that, in almost all animal species, except for mice (Cadet *et al.*, 1995), MDMA toxic effects appear to involve 5-HT systems. There is also molecular evidence for the involvement of a number of metabolic pathways in MDMA-induced neurotoxic damage to the brain. Using techniques of microarray analyses, it has been shown that MDMA administration influences the expression of several genes that code for proteins that are involved in metabolism and stress responses (Thiriet *et al.*, 2002). These changes in expression include increases in mRNA levels for Gpx-1 and heme oxygenase (Thiriet *et al.*, 2002). Because MDMA is metabolized via pathways that can induce the formation of superoxides and peroxides via redox-cycling (Cadet *et al.*, 1994b; 1995, Buchert *et al.*, 2001), the changes in these enzymes might constitute compensatory responses to incipient oxidative damage. A schematic diagram of MDMA-induced events that might cause degeneration of 5-HT terminals is presented in Fig. 3.

Possible Role of Glutamate and Nitric Oxide in MDMA-induced Toxicity

Glutamate is a neurotransmitter that can cause cell death both *in vitro* and *in vivo* (Dawson and Dawson, 1998). It has been suggested that glutamate might also be involved in MDMA toxicity (Atlante *et al.*, 2001; Battaglia *et al.*, 2002; Stewart *et al.*, 2002). For example, blockade of NMDA receptors with the antagonist, MK-801, was able to provide some protection against MDMA-induced 5-HT depletion (Farfel *et al.*, 1992; Colado *et al.*, 1993; Atlante *et al.*, 2001; Battaglia *et al.*, 2002; Stewart *et al.*, 2002), although MK-801 had no effect on drug-related decreases in TPH activity (Johnson *et al.*, 1989). The role of NO in MDMA toxicity also has been investigated in rats. It has been reported that NG-nitro-L-arginine methyl ester (L-NAME), an inhibitor of NO synthase, protects against the neurotoxic effects of MDMA via a mechanism that involves temperature regulation *in vivo* (Taraska and Finnegan, 1997).

Role of Hyperthermia in MDMA Neurotoxicity

The amphetamines, including MDMA, are known to cause hyperthermic responses (Nash *et al.*, 1988; Gordon *et al.*, 1991; Dafters, 1995; Dafters and Lynch, 1998). A number of drugs that attenuate MDMA toxicity also prevent the marked drug-induced hyperthermia. Specifically, 5-HT_2 receptor antagonists that block the hyperthermic response also protect from MDMA toxicity (Nash *et al.*, 1988). Morever, preventing the hypothermic responses produced by ketanserin also abolished its protective effects (Malberg *et al.*, 1996). In contrast, some agents, such as fluoxetine, that provide protection against MDMA neurotoxicity do not block the MDMA-induced temperature increase (Nash

et al., 1988; Mechan *et al.*, 2002). The evidence suggests that hyperthermia might be a member of a complex set of events that paprticipate in the toxic cascades caused by the drug.

It is interesting to note that interactions between the hypothalamic-pituitary-thyroid axis and sympathetic nervous system might be involved in MDMA-related hyperthermic responses (Sprague *et al.*, 2003). For example, removal of either the pituitary or thyroid glands was shown to prevent hyperthermia produced by drug treatment (Sprague *et al.*, 2003). In addition, the use of antagonists of $\alpha 1$ and $\beta 3$ adrenergic receptors was able to attenuate MDMA-induced temperature increase when used alone and could abolish the thermic response when the drugs were co-administered (Sprague *et al.*, 2003; 2005). Of further interest is the report that the skeletal muscle uncoupling mitochondrial protein 3 (UCP-3) is also involved in mediating MDMA-mediated hyperthermia because UCP-3-deficient mice treated with the drug showed blunted hyperthermic responses (Mills *et al.*, 2003).

CONCLUDING REMARKS

The amphetamines have a long history of illicit use among the various classes in societies around the world. The abuse of these drugs has continued unabated inspite of the documentation of the clinical and basic toxicology. In this review, we have presented evidence that oxidative and excitotoxic mechanisms, hyperthermic responses, and other metabolic processes are involved in causing the neurodegenerative effects of AMPH, METH and MDMA. In addition, both AMPH and METH have now been shown to cause cell death in various regions of the rodent brain via mechanisms that involve mitochondrial pathways. Moreover, METH-induced neuronal apoptosis appears to also be dependent on the activation of caspase-12 through the endoplasmic reticulum (ER) death pathway. More recently the Fas/FaL receptor-mediated cell death mechanisms were also shown to be involved in METH toxicity. Microarray analyses have also documented the involvement of molecular pathways that were not initially thought to participate in mediating the effects of these drugs. Thus, modern neurobiological techniques are offering more information on the nefarious effects of these drugs. It is hoped that this review will provide a substratum for other investigators to build upon.

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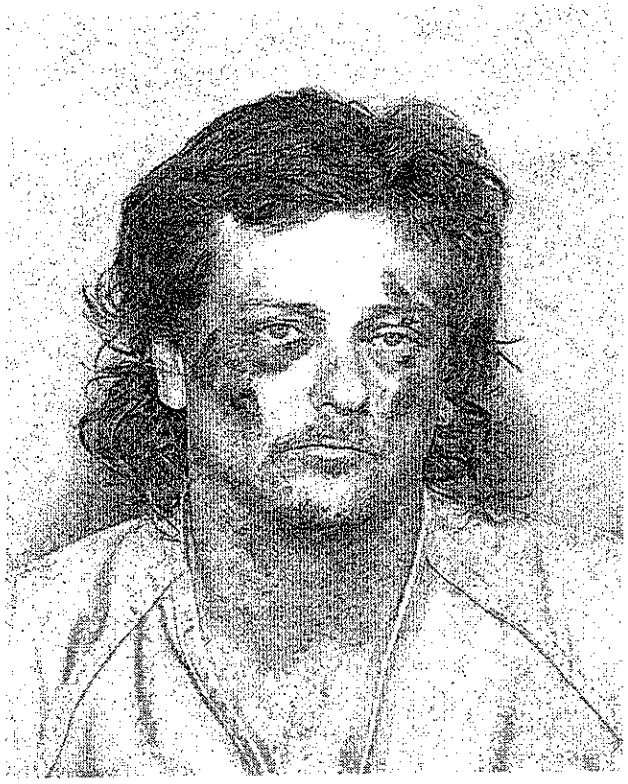
Appendix H

#1



DO YOU BELIEVE HIM?

#2

WHY SHOULD YOU BELIEVE ANYTHING HE SAYS ABOUT THE ASSAULT?

#3

ASSAULT 1°

EVIDENCE

+

COMMON SENSE

+

LAW

GUILTY

#4

ATTEMPTED ROBBERY 1°

INTENT TO COMMIT ROBBERY 1

+

SUBSTANTIAL STEP

GUILTY

#5

KIDNAPPING 1°

Intentionally abducted Angel Benson

+

With intent to use as shield or hostage

GUILTY

#6

COUNT IV: OBSTRUCTING

GUILTY

#7

ATTEMPTED ROBBERY 2°

INTENT TO COMMIT ROBBERY 2

+

SUBSTANTIAL STEP

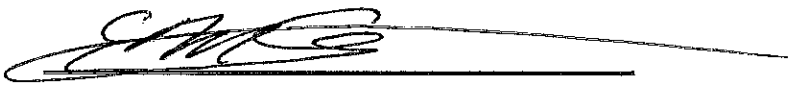
GUILTY

#8

#4

#10

I declare under the penalty of perjury that I am the petitioner and that the foregoing is true and accurate to the best of my knowledge.



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RE: In re PRP of Edward M. Glasmann ~ No. 39700-5

Clerk of the Court:

Enclosed please find an original and a copy of a *Personal Restraint Petition* for filing. I have also included a check for the filing fee in the amount of $250. Please let me know if I can provide additional information.

Sincerely,

Jeffrey E. Ellis
Attorney at Law